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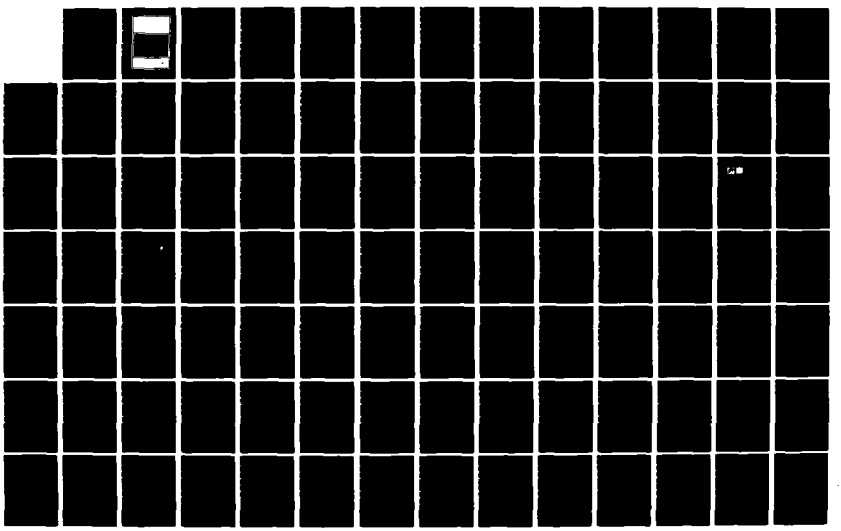

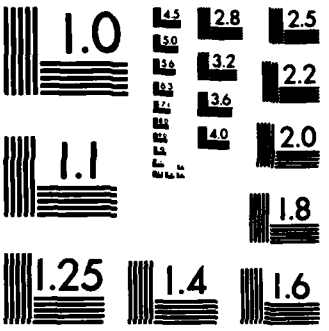

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AGARD LECTURE SERIES No.138

The Impact of Proposed Radio Frequency Radiation Standards on Military Operations

NORTH ATLANTIC TREATY ORGANIZATION

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ADVISORY GROUP FOR AEROSPACE RESEARCH AND DEVELOPMENT

(ORGANISATION DU TRAITE DE L'ATLANTIQUE NORD)

AGARD Lecture Series No.138

THE IMPACT OF PROPOSED RADIO FREQUENCY RADIATION STANDARDS ON MILITARY OPERATIONS

The material in this publication was assembled to support a Lecture Series under the sponsorship of the Aerospace Medical Panel and the Consultant and Exchange Programme of AGARD presented on 11—12 April 1985 in Rome, Italy, 15—16 April 1985 in Lisbon, Portugal and 18—19 April in Paris, France.

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Published March 1985

ISBN 92-835-1494-7

Printed by Specialised Printing Services Limited
40 Chigwell Lane, Loughton, Essex IG10 3TZ

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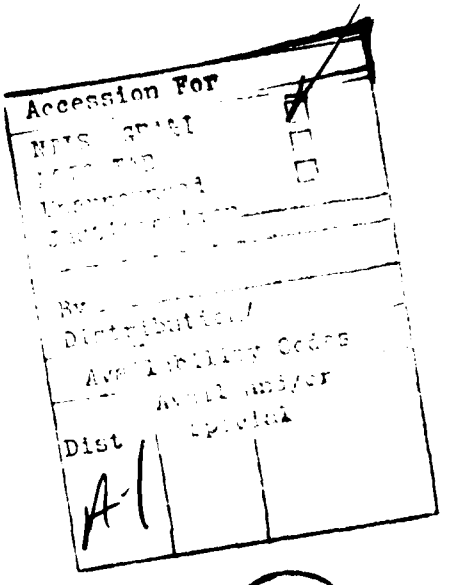

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THE IMPACT OF PROPOSED RADIOFREQUENCY RADIATION STANDARDS ON MILITARY OPERATIONS

by

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OVERVIEW

The development and application of devices that emit radiofrequency radiation (RFR) have significantly increased the quality of life throughout the world. Yet in recent years the beneficial aspects of radiofrequency/microwave technology have been somewhat overshadowed by a public fear of potential biological effects. This fear, in turn, has increased RFR research, led to a much better understanding of the interactions of RFR fields and biological systems, and resulted in the promulgation of new RFR safety guidelines.

Radiofrequency radiation is generally identified as nonionizing electromagnetic emission in the frequency range from 10 kHz to 300 GHz. Systems and devices which emit such radiation include radio and television broadcast transmitters, microwave ovens, RF sealers, RFR diathermy, and radars (military and civilian, mobile and fixed base). In the military, personnel work around active RFR emitters ranging from very small, low-power units to relatively high-power systems. RFR exposures can result from normal operations or from maintenance and calibration procedures. For example, a typical aircraft radar, energized on the ground, can produce average power densities of 10 - 20 mW/cm^2 at distances of 3 - 6 meters from the antenna, and accidental exposures at average power density levels of 100 -1000 mW/cm^2 have occurred.

Many mobile radio units, including citizen band (CB) transmitters, produce power density levels of 20 - 25 mW/cm^2 within 15 cm of the antenna, but less than 1 mW/cm^2 at distances of 2 meters or more. RFR exposures may range from 0.001 mW/cm^2 to 10 mW/cm^2 when the system is keyed, depending on the installation arrangement and vehicle. Similar exposures are possible from many "walkie talkie" units with the short coiled antenna, i.e., 10 mW/cm^2 within 10 cm of the antenna and less than 1 mW/cm^2 at a distance of 25 cm.

For more than 20 years most of the free world have used a single value to maintain personnel safety for radiofrequency radiation exposure. A power density of 10 mW/cm^2 time averaged over any 6-minute period has been applied as an acceptable exposure level, and it was generally thought to include a safety factor of ten. More recently, it has become clear that the inherent risks to health from RFR exposures are directly linked to the absorption and distribution of RFR energy in the body, and the absorption and distribution are strongly dependent on the frequency of the incident radiation. Therefore, in 1982, when new RFR safety guidelines began to emerge, a frequency dependent concept was incorporated. This resulted in permissible average power density levels for occupational environments that, depending on frequency, were 2 to 10 times lower than those previously used, and which may have significant impact on future operations.

This lecture series includes presentations on (1) the physical interactions of RFR fields with biological systems, (2) the biological effects of RFR exposures, (3) the procedures for measuring RFR fields in military operations, and (4) the development and operational impact of new RFR safety guidelines.

THE PHYSICAL INTERACTIONS OF RADIOFREQUENCY RADIATION FIELDS AND BIOLOGICAL SYSTEMS

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SUMMARY

A biological system irradiated by radiofrequency radiation (RFR) responds to the internal RFR fields produced by that irradiation. The measurement and calculation of the internal fields is called dosimetry. The internal fields are often described in terms of the specific absorption rate (SAR) in watts/kilogram. SARs are usually determined experimentally by measuring temperature rise in the absorber (either by discrete probes or thermographic cameras) or by directly measuring the internal electric field. A combination of techniques, each valid for a particular model and in a particular frequency range, have been used to calculate average whole-body SARs for models of human beings and other animals over a wide frequency range for plane-wave irradiation. Calculating SARs for near-field irradiation is much more difficult than for plane-wave irradiation; thus fewer near-field SAR data are available. To calculate the spatial distribution of SARs is still more difficult (especially at higher frequencies); this problem in dosimetry is yet to be solved satisfactorily, although significant progress has recently been made in this area.

INTRODUCTION

The ever-increasing radiofrequency (RF) electromagnetic equipment and the resulting increased exposure of both occupational workers and the general public to RF radiation (RFR) has caused growing concern in recent years about possible health hazards of RFR exposure. (RF is defined as the frequency range from 10 kHz to 300 GHz.) The study of how electromagnetic fields interact physically with biological systems is an essential part of research related to biological effects produced by exposure. Dosimetry, the calculation and measurement of the internal RF fields produced in an object exposed to RFR, is a principal component in this study.

Dosimetry is very important because biological effects are related to the internal fields in the body, which are not the same as the incident fields of the radiation. Determination of the internal fields, either by calculation or measurement, is often very difficult. Generally speaking, the internal fields are a function of the incident fields, the size and shape of the object, the electrical properties of the object, and the frequency of the incident fields. Thus for a given incident radiation field, the internal fields in one object may be quite different from the internal fields in another object.

The purpose of this paper is to describe in general terms the common theoretical and experimental methods used in dosimetry, along with some results and a summary of what can be done at this time. The paper is written primarily for those who are not electrical engineers or physicists; therefore only a minimum of mathematical details is included. No exhaustive description of what is to be found in the literature on dosimetry is given and no attempt has been made to be all inclusive. Only RFR dosimetry as applied to models of people and animals is treated. This paper is similar to one in the proceedings of the previous radiofrequency radiation workshop, conducted by Research Study Group 2 of NATO AC/243 Defence Research Group Panel VIII (published as Aeromedical Review, USAFSAM Review 3-81, September 1981, by the U.S. Air Force). For convenience of the reader, part of the background material in that paper is included here.

In determining the internal RF fields inside an irradiated object, both theory and experiment are needed. The theory is needed to explain how the internal fields depend on the characteristics of the incident fields and the absorber, to show cause-and-effect relationships, and to provide ways to predict the internal fields for a given set of conditions. Theoretical methods are also needed to allow extrapolation of observed RFR-related biological effects in animals to effects expected to occur in man; for obvious reasons of safety, most experiments for studying RFR-related biological effects cannot be performed directly on people.

Experimental methods are needed to verify theory, to provide additional understanding of the nature of internal fields, and to obtain data for cases in which theoretical calculations cannot be made. Researchers traditionally use a combination of theoretical and experimental techniques to learn as much as possible about dosimetry.

Theoretical methods consist, in one way or another, of solving Maxwell's equations, the fundamental basis of electromagnetic theory, for the particular absorber and radiation fields of interest. This includes representing both the actual absorber (usually an animal or a person) and the incident radiation fields by mathematical models. The mathematical models are never completely accurate representations of either the absorber or the incident fields, so the calculated internal fields are only approximations to any real physical values. The better the model is, the more complicated the calculations usually are.

Experimental methods typically measure either the internal electric field or the temperature rise at internal points. Under certain conditions, both the internal fields and the energy absorbed can be calculated from the internal temperature rise.

Next, some basics of electromagnetics are described; then theoretical methods are discussed in two groups : analytical and numerical. Work in near-field dosimetry is described next, followed by some experimental results. Finally, some qualitative explanations of absorption are given; then a summary.

SOME BASICS OF ELECTROMAGNETICS

The framework of electromagnetics is Maxwell's equations, which describe the relations between the electric ($\underline{E}$) field, the magnetic ($\underline{H}$) field, and the sources (charge and current) that produce these fields. Other auxiliary equations describe the interaction of these fields with materials. Since Maxwell's equations are very difficult to solve, a variety of special techniques have been used to solve them for special conditions. For example, when the frequency of the radiation is low enough that the wavelength is very long compared to the size of the objects, Maxwell's equations may be approximated by the equations of circuit theory. For extremely high frequencies where the wavelength is very small compared to the size of the objects, Maxwell's equations are approximated by the equations of optics. In the range where the wavelength is about the same size as the object, the techniques of microwave theory apply.

Likewise, in dosimetry a combination of techniques and models has been used. As a prelude to the description of these techniques in the next section, some fundamentals of electromagnetics are explained in this section.

Radiation Fields

Radiation fields are fields to which the object is exposed and which would be measured in the absence of the object. Radiation fields are usually categorized according to frequency, the magnitudes of $\underline{E}$ and $\underline{H}$, and their spatial variation, as described below.

There are two general classes of radiation fields: near fields and far fields. The mathematical expressions for EM fields contain terms like $1/r$, $1/r^2$, $1/r^3$, ..., where r is the distance from the source. In a region "far" from the source, the terms $1/r^2$, $1/r^3$, ... become negligible compared to the $1/r$ term, and the fields are said to be far fields. When the higher-order terms in r cannot be neglected, the fields are called near fields. The near fields vary more rapidly with distance than the far fields and are generally more difficult to handle mathematically. The far fields are often described as propagating waves.

One common type of far-field wave is a spherical wave, in which the wavefronts form spheres. If the radius of the spherical wavefront is large enough, the spherical wave approximates a plane wave. A plane wave is a mathematical model; it does not occur physically. In plane waves, the wavefronts are planes and the magnitudes of both E and H are constant throughout a given plane, which, of course, is not physically possible. The plane wave, however, is a very useful model because it is relatively simple mathematically and it does form a useful approximation to some far fields. The plane wave has been widely used in dosimetry to provide important understanding as well as approximate results.

The defining characteristics of a plane wave are--

1. The wavefronts are planar.

2. $\underline{E}$ and $\underline{H}$ are perpendicular to each other and are both perpendicular to the direction of propagation.

3. In free space, $E/H = 377$ ohms, which is called the wave impedance.

Another important feature of plane waves is that the radiation fields can be completely specified by (1) the orientation of the $\underline{E}$ field (or the $\underline{H}$ field), (2) the direction of propagation, and (3) the power density. The time-average power density in a plane wave is given by

$$P = EH = E^2/377 \quad (\text{in } W/m^2 \text{ if E is in RMS V/m})$$

where E and H are the magnitudes of the electric and magnetic field vectors, respectively.

Absorption Characteristics

Material Properties--Electric fields transfer energy to material bodies by three principal mechanisms:

1. $\underline{E}$ fields give kinetic energy to electrons that are not tightly bound to any one atom. These are called free electrons.

2. $\underline{E}$ fields induce electric dipoles in atoms and molecules. This is called polarization. The "friction" associated with polarization results in "heating" the material, which represents a transfer of energy to the material from the $\underline{E}$ field.

3. $\underline{E}$ fields align electric dipoles already existing in the material. The "friction" associated with this alignment results in an energy transfer to the material.

These three energy-transfer mechanisms (also often called loss mechanisms because they represent energy lost from the $\underline{E}$ field to heat the body) are traditionally described by material properties called permittivity and conductivity. For EM fields that vary sinusoidally with time, one property, complex permittivity, describes all the loss mechanisms. The complex permittivity is given by

$$\varepsilon = \varepsilon_o(\varepsilon' - j\varepsilon'') \quad (1)$$

where ε_o is a constant called the permittivity of free space; ε' is the real part of the relative complex permittivity, or dielectric constant; ε'' is the imaginary part; and $j = \sqrt{-1}$. The ratio $\varepsilon''/\varepsilon'$ is called the loss tangent. Many tables list both ε' and the loss tangent. Other tables list only ε' and σ, the dc conductivity, which is related to ε'' by $\sigma = \omega\varepsilon_o\varepsilon''$, where ω is the radian frequency.

Specific Absorption Rate (SAR)--The rate of energy transfer to a material is commonly described in dosimetry by the specific absorption rate (SAR). The SAR is defined as the time rate of energy transfer to the body, per unit mass. The average SAR is the total energy transferred to the body per unit time divided by the total mass of the body. The local SAR is the rate of energy transferred to an infinitesimal volume at a point in the body, divided by the mass of the infinitesimal volume. For sinusoidal fields, the SAR at a given internal point is:

$$\text{SAR} = \frac{1}{\rho}\,\omega\varepsilon_o\varepsilon''E_{in}^2 \text{ watts/kilogram} \quad (2)$$

where

ρ is the mass density in kilograms/meter^3.

ε_o is the permittivity of free space in farads/meter.

ε'' is the imaginary part of the relative complex permittivity.

ω is the radian frequency. ($\omega = 2\pi f$, where f is the frequency in Hertz.)

E_{in} is the magnitude of the internal electric field at the point in RMS volts/meter. The internal field is not equal to the incident field.

The SAR is the mass normalized rate of energy transfer, so it is equivalent to the mass normalized power transferred to the body, commonly called absorbed power density. Since heat generated in a body is directly proportional to the absorbed power, the SAR is often of major interest in dosimetry. It is important to note, however, that the temperature of the body is not necessarily proportional to the SAR, since temperature is the result of all the thermal properties of the body in addition to the SAR. Although regions of intense localized SAR are sometimes referred to as "hot spots," this nomenclature is not precise because the temperature may or may not be correspondingly high at that point, depending on the heat transfer characteristics of the body.

From equation (2), we can see that for a given E_{in} and ω, the SAR is directly proportional to ε''. Thus, a body with a higher ε'' is said to be more absorbing, or more lossy, than a body with a lower ε''. Generally speaking, ε'' tends to be higher for "wetter" materials and lower for "drier" materials. For example, the ε'' for dry paper is very low, while that for wet paper is relatively high. Wet paper put in a microwave oven will heat up until it dries out and then will no longer heat. In biological materials, bone and fat are not as lossy as muscle.

Penetration and Frequency Characteristics--In dosimetry, an important characteristic is the dependence of absorption on frequency. As you might expect, this frequency dependence is very complex, but some simple characteristics provide adequate qualitative understanding of dosimetry. The depth of RFR penetration in a lossy medium is a strong function of both frequency and permittivity. For a given permittivity, low-frequency radiation penetrates more deeply than high-frequency; thus high-frequency radiation characteristically produces only surface heating. At a given frequency, RFR penetrates deeper into materials of low permittivity than those of high permittivity. Figure 1 can make these two characteristics clearer; it shows the skin depth of a plane wave incident on a planar dielectric half space, as a function of frequency. Skin depth is defined as the distance at which the EM fields have reduced to e^{-1} (0.368) of their value at the surface. This corresponds to the power absorption being e^{-2} (0.135) of the surface value. The plot in Fig. 1 is for material with a permittivity two-thirds that of muscle, which is an average permittivity for the human body. Since the skin depth depends on both ε' and ε'', and since both ε' and ε'' increase as the frequency decreases, the skin depth does not increase as fast when the frequency decreases as it would if the permittivity did not depend on frequency.

Since the curve in Fig. 1 is for a planar dielectric half space, it does not tell much about the penetration depth in man-sized models at lower frequencies; but it does give important qualitative information.

Polarizaton--An important factor in dosimetry is polarization, which is the orientation of the EM field vectors with respect to the body. For plane-wave fields the polarization is designated by which of vectors $\underline{E}$, $\underline{H}$, or $\underline{k}$ is parallel to the long axis of the body, where $\underline{k}$ is a vector in the direction of propagation. If the $\underline{E}$ field is parallel to the long axis of the body, the polarization is called E polarization. Similarly, H and K polarizations are when $\underline{H}$ and $\underline{k}$, respectively, are parallel to the long axis of the body.

These three definitions of polarization are sufficient for bodies of revolution about the long axis, such as a cylinder and a prolate spheroid (like an egg). The human body, however, is not a body of revolution; hence more detailed definitions of polarization have been made [1] but are not necessary for discussion in this paper. Polarization is important because the SAR depends strongly upon it, as explained in the section QUALITATIVE EXPLANATIONS OF ABSORPTION CHARACTERISTICS.

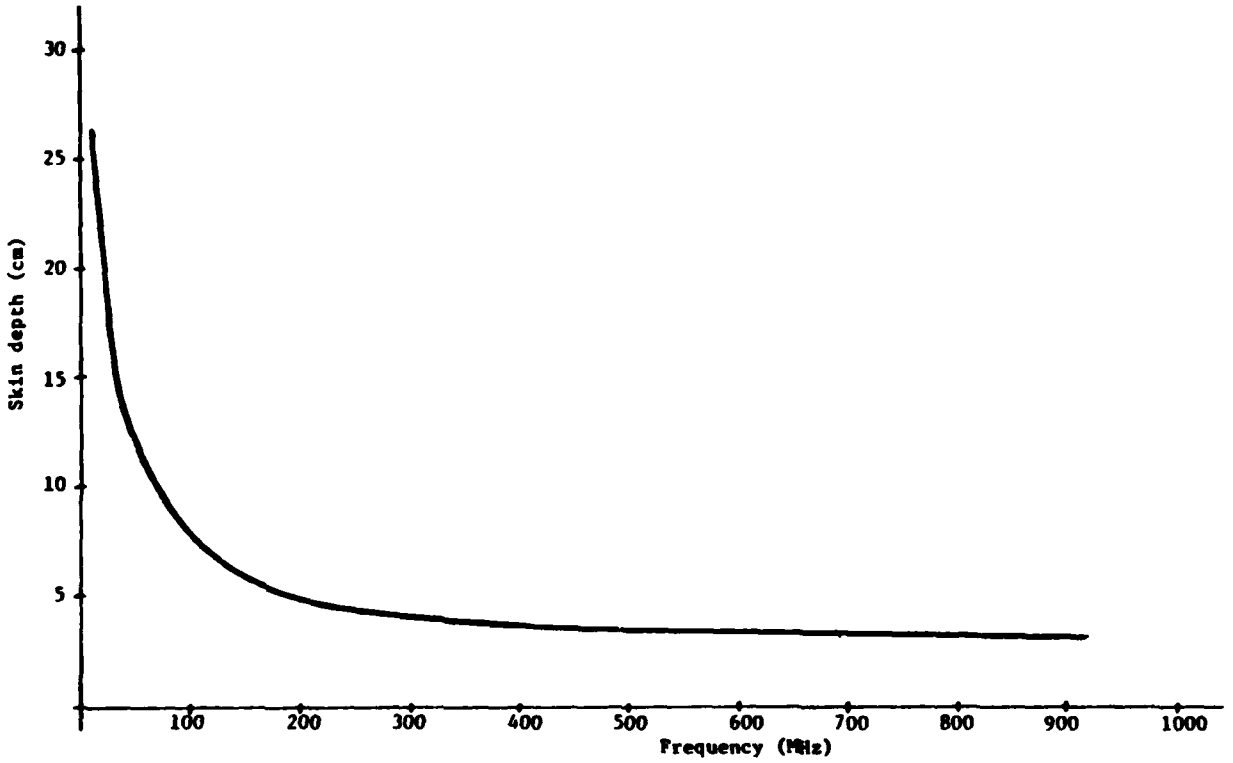


Fig. 1. Skin depth as a function of frequency for an EM plane wave incident on a dielectric half space having a permittivity two-thirds that of muscle tissue.

Measurements

Two basic techniques are used to measure internal fields. One is to use an E-field probe designed for use inside an object. These probes provide a direct measurement of E_{in}, which can then be used in equation (2) to calculate the SAR if the value of ϵ'' is known. The main difficulty with internal E-field probes is getting enough sensitivity in a small enough probe. The problem is especially difficult at the lower frequencies, where the wavelength is so long that a physically small probe is very short compared to a wavelength and consequently is insensitive. Another problem is the difficulty of designing a probe so that it reads independently of the permittivity of the material in which it is placed. Some good probes have been designed for use in biological material, however, and better ones are being developed.

A second basic method is the measurement of temperature rise in the body. Johnson and Guy [2] showed that if the incident radiation is strong enough to produce a temperature rise linear with time, the SAR can be calculated from the temperature increase without knowing the detailed heat transfer characteristics of the body. This is possible because thermal diffusion is negligible if the temperature rise is linear. The equation they derived is:

$$SAR = \frac{4.186\ \rho c\ \Delta T}{\Delta t}$$

where

SAR is in W/cm^3.

ρ is the mass density in g/cm^3.

c is the specific heat of the tissue in cal/g°C.

ΔT is the temperature change in °C.

Δt is the time of exposure in seconds.

Several temperature probes that will not perturb internal fields have been developed and can be used to measure temperature rise during RFR. Whole-body calorimetry can also be used to measure the average SAR in small animals.

Figure 2 shows the average SAR as a function of frequency for models of an average man irradiated

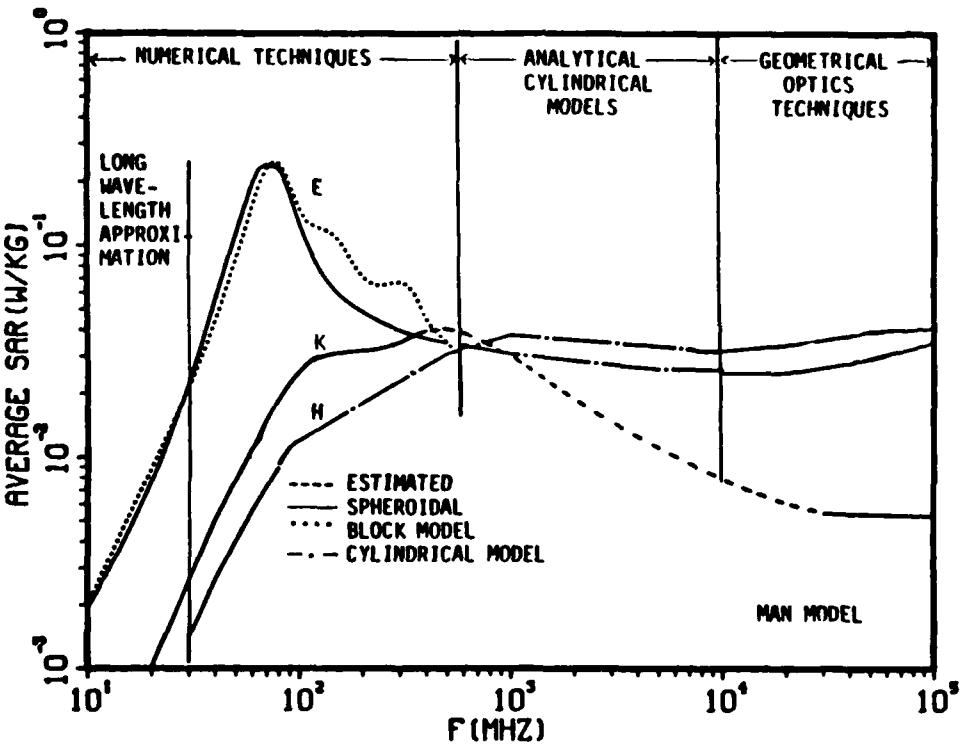


Fig. 2. Average SAR for models of an average man irradiated by an EM plane wave of 1 mW/cm^2 power density. E, K, and H designate polarizations in which the incident electric field vector, propagation vector, and magnetic field vector, respectively, are parallel to the long axis of the body. The various methods used to make the calculations are shown.

by an EM plane wave for the three polarizations. A combination of techniques has been used in theoretical dosimetry calculations because the problems are so complex that no one technique by itself is adequate. Extremely important information about the absorption characteristics of humans and other animals has been obtained by combining results from several techniques. As shown in Fig. 2, several kinds of models have been used, as well as several methods of computation.

Numerical techniques have been used for frequencies up to about 600 MHz. Beyond this frequency, numerical techniques have not been used because they require excessive amounts of computer storage. Numerical techniques consist of complex computer programs that require large amounts of computer storage and long run times. Numerical techniques are often characterized by the solution of large matrix equations obtained either from a discrete form of Maxwell's equations or from simultaneous equations for coefficients of series solutions. In the matrix equation obtained from the Green's function integral equation, a matrix element corresponds to the field intensity in a mathematical cell in the body. Since the field intensity in each cell is often assumed to be constant throughout the volume of the cell, the mathematical cells must be smaller at high frequencies where the wavelength is smaller and the spatial variation in the fields correspondingly more rapid. Thus, at high frequencies, many mathematical cells are required, making the matrices large and the matrix inversion very difficult.

In the lower frequency range, an analytical approximate solution of Maxwell's equations is useful. This approximation is valid when the wavelength of the incident radiation is large compared to the size of the body, which occurs up to frequencies of about 30 MHz for man-sized models. The technique is limited to spheroidal and ellipsoidal models, which are not good representations of the human body in the sense that they do not represent shapes such as those of arms and legs well enough. Therefore the method gives little useful information about local SAR, although it has furnished useful information about average SAR. As shown in Fig. 3, the average SAR in spheroidal models is nearly the same as that in block models [3]. This is very fortuitous because the approximate methods are much easier to use than the more complicated numerical techniques. Numerical techniques, however, can furnish much better information about local SAR.

For frequencies beyond 400 MHz, two techniques have been primarily used for calculating the SAR. One is the analytical solution of Maxwell's equations for cylindrical models. This technique is useful when the wavelength is short compared to the length of the body, which occurs above about 400 MHz for man-sized models. Because of computational difficulties, it can be used only up to about 7 GHz. Above 7 GHz another approximation, based on geometrical optics techniques, is useful. In this approximation, the wavelength is assumed to be very short compared to the size of the body, which allows the incident radiation to be described by rays. An additional approximation is made that the internal absorption is high

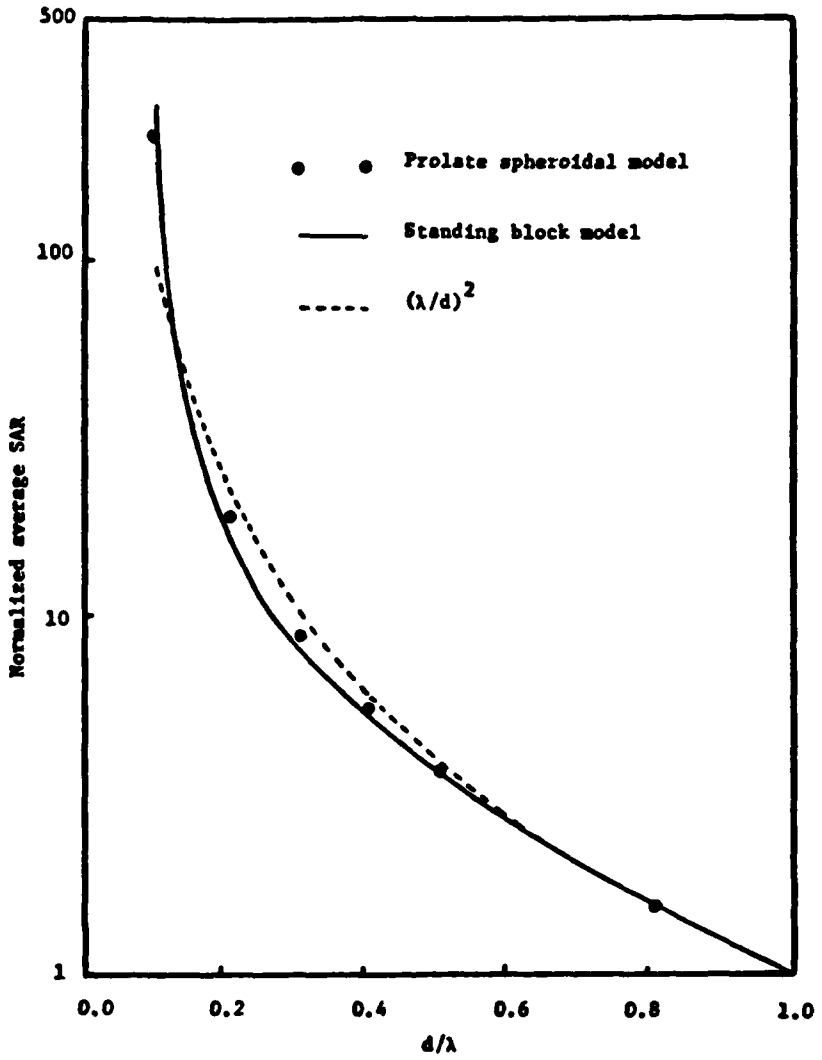


Fig. 3. Normalized average SAR in spheroidal models compared to block models [3], both irradiated by a short electric dipole. d = distance from the dipole; λ = wavelength.

enough for the rays not to be internally reflected, but completely absorbed.

Qualitative explanations for the characteristics of the curves shown in Fig. 2 are given in the section QUALITATIVE EXPLANATIONS OF ABSORPTION CHARACTERISTICS. The theoretical methods used to obtain the curves in Fig. 2 will now be described in more detail in two main categories: basic analytical and numerical techniques commonly used in electromagnetic dosimetry. Then developments in dosimetry that have occurred in the last 3 or 4 years are discussed.

Analytical Techniques

Analytical techniques are so called because, in contrast to numerical techniques, they consist of some solution to Maxwell's equations that is not based on a direct numerical solution and does not require the inversion of large matrices.

Planar and Spherical Models--Because of their mathematical simplicity, planar and spherical models were used in early work in calculating the SAR [4-6]. Planar models, of course, are the simplest mathematically. However, the information obtained from planar model analyses is useful mostly for some qualitative understanding; the data do not represent absorption by human bodies very well. The sphere [6], although a better model, is still quite limited in representing the shape of the human body and is more difficult to analyze than planar models.

Although the analyses of planar and spherical models were very limited in terms of providing useful data, they did provide important first steps in understanding theoretical dosimetry. The spherical model [6] showed a resonance similar to that of the K polarization in Fig. 2, but naturally could not show the polarization effects. Analyses of multilayered spherical models [7] have shown that the resonance properties are significantly different for the layered models than for the homogeneous. The multilayered spherical models have primarily been used for investigating absorption in the human head.

Long-Wavelength Analyses--Extensive use has been made of an approximation with spheroidal and ellipsoidal models that is valid when the wavelength is long compared to the size of the model [8, 9].

In this method, Maxwell's equations are expanded in a power series in k, the free-space propagation constant. Approximations are then made to obtain simpler equations that are valid in the low-frequency range. These are easier to solve than Maxwell's equations because they require only solutions to Laplace's equation instead of the solution to wave equations. The approximation is valid for man-sized models up to about 30 MHz.

Since these spheroidal models do not include features such as arms and legs, the analysis does not provide much useful information about SAR distribution; however, much useful information about average SAR has been obtained from these approximate calculations. For example, the polarization effects shown in Fig. 1 were first calculated by the long-wavelength approximation for prolate spheroids. The long-wavelength analyses provide an easy way to calculate the SAR at low frequencies for any size spheroid, provide important insight into the qualitative nature of SAR characteristics, and provide a good check for numerical techniques at lower frequencies.

Cylindrical Models--At first, average SARs in the range of frequencies from about 400 MHz to 7 GHz were difficult to calculate. In this range, numerical techniques cannot be used because the wavelength is short enough that the matrices are extremely large and difficult to invert, yet the frequency is still low enough that the short-wavelength approximations are not valid. In this frequency range, however, cylindrical models provide useful information [10].

As shown in Fig. 2, the results from the cylindrical model agree well with those from other models in the transition region near 400 MHz. In the cylindrical model, standard electromagnetic techniques based on classical electromagnetic equations are used. Although the solution is well known and straightforward, the technique is limited at the high end of the frequency range by difficulties in calculating the higher-order Bessel functions of large complex argument that occur in this range.

Since the cylindrical model is infinitely long, it cannot be used for K polarization; however, it has provided much useful information for E polarization and H polarization.

Spheroidal Wave Functions--Another analytical technique that has been used is the direct solution of Maxwell's equations in spheroidal coordinates [11]. Although a formal solution has been obtained, the numerical calculation of the SAR has been so difficult that little useful information has been obtained for RFR dosimetry in human models.

Empirical Techniques--Several empirical relations for describing the SAR have been developed [12, 13]. These relations are based on the characteristic behavior of the SAR for E polarization as shown in Fig. 2, and they were obtained simply by finding equations that would reproduce the SAR-versus-frequency curves. Durney et al. [14] did this by obtaining the least-squares best fit of the relation to all the data calculated for prolate spheroidal models of humans and animals, as given in the second edition of the Radiofrequency Radiation Dosimetry Handbook [1]. The result is a simple formula that can be used to calculate the SAR for E polarization as a function of frequency for prolate spheroidal models ranging in size from rats to humans. The equation is simple enough to be used with a hand calculator, and the accuracy is a few percent.

Similar techniques have been used to obtain information for models of humans standing on or near ground planes [15], such as a man wearing shoes and standing on perfectly conducting ground. The main effect of the ground plane is to shift the resonant frequency.

Numerical Techniques

The following numerical techniques have been commonly used in electromagnetic dosimetry.

Moment Method--A numerical technique called the moment method has been used to calculate both average and local SARs in block models of man [16-19]. This method is based on the solution of an integral equation in which the electric field in each mathematical cell of the model is represented by a pulse function (the electric field has a constant value everywhere inside the mathematical cell). Several block models have been used with this method. A 180-cell block model used by Hagmann et al. is shown in Fig. 4. It was designed by choosing the cubical cells to form the volume and shape most closely resembling an average man.

Very useful data have been obtained by the moment method, with calculations ranging up to 600 MHz for man-sized models. Beyond 600 MHz the method is not practical because it requires too much computer memory.

Extended Boundary Condition Method--The extended boundary condition method (EBCM) differs from the moment method in that the EBCM makes use of a spherical harmonic expansion of the incident and scattered EM fields [20, 21]. A system of linear equations relating the unknown expansion coefficients of the scattered field to the known coefficients of the incident field is obtained from the boundary conditions and solved by matrix inversion. For man-sized models, the EBCM can be used to obtain SAR data up to about 70 MHz for E polarization.

Some Recent Developments

This section summarizes some of the work done in the last 3 or 4 years. Work described in the previous sections was done prior to that time; much of it in the 1970s.

Moment Method Using Smaller Cells--The use of pulse functions in the moment-method solution was shown by Massoudi et al. to result in values for the local distribution of SAR that are of limited accuracy [22]. They showed this by dividing each cubical mathematical cell into smaller cells and recalculating the local SARs. The values for the local SARs did not converge as the size of the cells was made

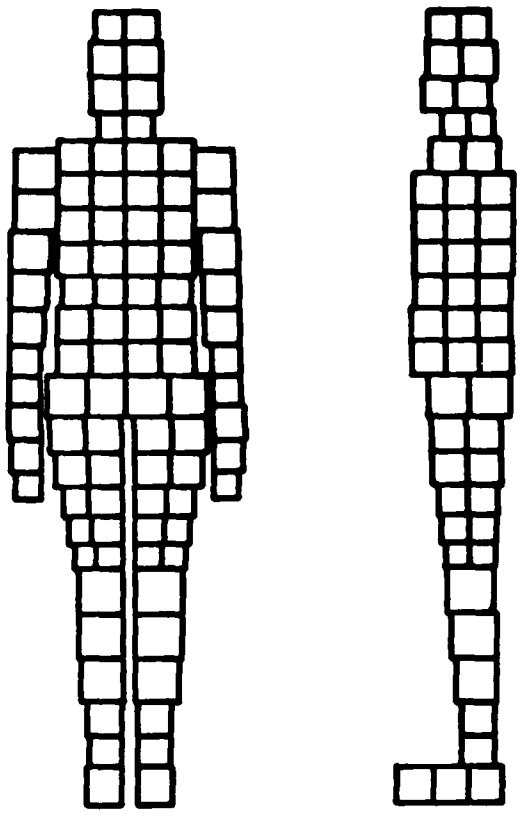


Fig. 4. A 180-cell block model of man used by Hagmann, et al. [18].

smaller. The reason seems to be that the pulse functions are not adequate approximations to the electric field in each cell because they cannot satisfy the boundary conditions between cells. The whole-body average SAR, on the other hand, did not change appreciably as the cells were subdivided, seeming to indicate that the pulse functions may give acceptable results for whole-body average SARs. However, the results obtained by DeFord et al. [23] for average SARs in block models having much smaller cells may indicate that calculations of average SARs using pulse functions are not very accurate.

DeFord et al. used the band approximation method for inverting large matrices to calculate SARs in block models having as high as 1132 cells, which is a significantly larger number of cells than the 180 cells typically used previously (Fig. 4) and requires a significantly greater amount of computer memory and processing time. For whole-body average SAR, their results are about 50% higher for the 1132-cell model than for the 180-cell model, both for homogeneous and inhomogeneous models. Also, the whole-body average SAR for the 1132-cell inhomogeneous model is about twice that for the 180-cell homogeneous model. This trend seems to be consistent with recent experimental results (see the section SOME RESULTS).

Borup and Gandhi [24] used fast-Fourier transform (FFT) methods with the moment method to avoid inversion of large matrices. This technique, since it requires significantly less storage in computer memory for a given system of equations, allows calculations with a much larger number of mathematical cells than would be possible using the traditional moment method with matrix inversion. At present, the FFT method has been used only for calculations in a two-dimensional model. It is not clear whether the method will be practical for three-dimensional calculations.

Moment Method Using Linear Basis Functions--Since the pulse functions used with the moment-method solution of the integral equations limit the accuracy of the solution, an obvious improvement to try is the use of linear basis functions. Using linear basis functions would be like approximating a function in one dimension by $ax + b$ instead of just by the constant b, which would be like the approximation of a pulse function. Linear basis functions have been used by Schaubert et al. [25] and by Tsai et al. [26]. In both cases, tetrahedral cells were used instead of the cubical cells used in the block models with the pulse functions. The basis functions used by Schaubert et al. do not accurately represent a field with arbitrary linear variation, but only linear variation in a direction defined by the basis functions. The advantage of this formulation is having only one unknown for each face of the tetrahedronal cells. This provides greater modeling power than the cubical cells with pulse functions. That is, the total number of unknowns per volume element of cells is less with the tetrahedral cells than with cubical cells. For a given number of unknowns, therefore, the tetrahedral-cell model would represent an inhomogeneous object better than the cubical-cell model.

The formulation used by Tsai et al. represents a field with arbitrary linear variation, but it results in a greater number of unknowns per cell. Because of the linear representation, however, somewhat larger cells can be used. The linear approximation of Tsai et al. also gives a smoother approximation to the fields inside the cells than does that of Schaubert et al., which is like a stair-step representation. Both formulations explicitly take into account the surface charge density on the surfaces of

the cells, which the pulse-function cubical cell model does not. Both Schaubert et al. and Tsai et al. make calculations for homogeneous and layered spheres and compare them to the results obtained from the classical Mie solution. Although it is difficult to compare accuracies, the use of linear basis functions promises better calculations of local SAR distribution, but at the price of more complexity and more computer time and storage. Using linear basis functions for calculations in models of man will require very large computers.

Iterative Extended Boundary Condition Method--The EBCM described in the previous section has been useful for calculating the SAR in spheroidal models, but the EBCM does not work at frequencies beyond approximately 70 MHz for man models because the matrix equation becomes ill conditioned. The ill conditioning occurs because the EBCM represents the EM fields in terms of a spherical harmonic expansion, and this expansion does not fit an elongated object very well at the higher frequencies. Lakhtakia et al. [27] improved the EBCM by incorporating two new procedures. The first is use of a multiple--instead of one--spherical harmonic expansion. This amounts to dividing the interior of the irradiated object into a number of overlapping spherical subvolumes, expanding the EM fields in each spherical subvolume into a separate spherical harmonic series, and then connecting the expansions together by requiring the fields to be continuous in the overlapping regions. This procedure avoids ill conditioning because the spherical harmonics fit the spherical subregions better than elongated regions. The second new procedure is iteration that begins with an approximate solution--for example, the solution for a perfectly conducting spheroid--and then iterates to obtain the solution for the actual object. The EBCM with these two new features is called the iterative extended boundary condition method (IEBCM). The IEBCM can be used to calculate average SARs for spheroidal models of man up to frequencies of about 400 MHz, which is far greater than the 70 MHz to which the EBCM calculations are limited.

Spectral Iterative Technique--Kastner and Mittra [28] have developed a numerical method quite different from those described above. It is called the spectral iterative technique (SIT). The SIT is based on a two-dimensional Fourier transform technique in which the numerical fast-Fourier transform (FFT) is used to compute the Fourier transforms. In the SIT, the Fourier transform method for a single arbitrary planar dielectric slab is used repeatedly to compute the SAR for a three-dimensional model. Thus, a model of a man would consist of a number of thin planar slabs, each of appropriate size and shape. An important feature of the SIT is that it does not require matrix inversion, in contrast to other numerical methods such as moment methods. This enables the SIT to be used for electrically larger bodies, with a large number of unknowns on the order of 2000 or more. The method has apparently not been used for dosimetry in models of man.

Finite-Difference Time-Domain Calculations--Another technique recently applied to electromagnetic dosimetry in models of human beings and other animals is the finite difference solution of Maxwell's equations in the time domain. This technique approximates the differential form of Maxwell's equations in the time domain by difference equations and then solves these equations by stepping through time. Although this technique was used nearly 20 years ago in other applications [29], only recently has it been applied to electromagnetic dosimetry in models of human beings and other animals [30, 31]. This technique has the advantage of not requiring inversion of large matrices and therefore can be used for calculations in models of the human body composed of as many as 10,000 cells. However, a disadvantage of the method is that the electromagnetic fields in the space surrounding the body must also be calculated, which means that the surrounding space must also be modeled by mathematical cells. This greatly increases the number of cells for which calculations must be made, thus requiring a large amount of computer memory. At the present time, only preliminary dosimetric calculations have been made by this technique.

Very-Low-Frequency and Medium-Frequency Techniques--In the frequency range from 10 kHz to 3 MHz, which includes the very-low-frequency (VLF) and medium-frequency (MF) bands, the SARs are very low, even for relatively intense incident fields. Consequently, other factors such as electric shock and RF burn may be more important dosimetric parameters in these frequency bands. Measurements in the VLF and MF bands have recently been made by Guy and Chou [32], by Gandhi and Chatterjee [33], and by Kanai et al. [34].

The quasi-static approximation can be used in the VLF-MF bands because the wavelength is very long compared to the size of the absorbing body. Also, the internal fields in a living subject are small compared to the external fields, and the perturbed external fields and induced-charge density are independent of the permittivity of the body tissues. The induced surface charge produces internal currents that are a function of the permittivity, but the total conduction current through any cross section of the body is independent of the permittivity. Measurements in baboons have shown that equipotential planes occur perpendicular to the long axis of the body for E polarizaton, which means that the internal potential distribution can be predicted from the surface potential distribution. Guy and Chou [32] measured the surface potential distribution on volunteers while applying a harmless low-level VLF current of known magnitude through the body. From these and geometrical body measurements, body resistance and SARs were calculated for a variety of conditions, such as for exposure of a subject grounded, in free space, and with feet grounded and one hand contacting a large object like a vehicle.

NEAR-FIELD DOSIMETRY

As explained in the section Radiation Fields, plane-wave analyses are often used in electromagnetic dosimetry because the mathematics are simple in comparison to other cases. However, many real-life exposures of people to electromagnetic radiation occur in the near fields. Consequently, being able to calculate the SAR produced by near-field irradiation is very important. As the research progressed in theoretical dosimetry, plane-wave analyses were first developed, providing much useful information and data. Then the work was extended to include the much more complicated near-field analyses. To attempt the near-field analyses without having the basic information provided by the simpler plane-wave analyses upon which to build would have been very difficult.

In contrast to plane waves, near fields are much more complicated in the following respects: First, the near fields tend to vary more rapidly with space, as explained in the section Radiation Fields. Second, the near-field electric and magnetic field vectors are not necessarily perpendicular. Third, the ratio E/H in free space is not necessarily 377. In addition, near fields are not always conveniently characterized by waves; they are often more nonpropagating in nature and are therefore called "fringing fields" or "induction fields." Also, the absorbers in the near field may couple strongly to the electromagnetic source and change the radiation produced by the source. All these factors combine to make the mathematical formulation of near-field absorption much more difficult than that of plane-wave absorption.

Another important complicating factor is that the absorption produced by near fields cannot be conveniently normalized to the incident power density, as it can be for plane waves. The incident fields for far fields can be fully characterized by just two quantities: the incident power density and the orientation of the fields with respect to the absorber. For near fields, however, no such standardization is possible because the $\underline{E}$ and $\underline{H}$ vectors are not necessarily perpendicular, and no parameter corresponds to the convenient power density that is so easily specified for far fields. This makes near-field dosimetry much more difficult to generalize; calculations for each near-field source must be made separately. Comparing the absorption characteristics produced by one source with those of another is therefore difficult. On the brighter side though, it turns out that the same qualitative explanations of absorption apply for both near fields and far fields, as explained later. A compilation of near-field dosimetric techniques and data is contained in the third edition of the Radiofrequency Radiation Dosimetry Handbook [35].

The work in near-field theoretical dosimetry began, as you would expect, with simple models and simple sources. The SAR produced in spheroidal and cylindrical models irradiated by the near fields of simple sources, such as short electric dipoles and small magnetic dipoles, have been calculated using the long-wavelength approximation explained in the section Long-Wavelength Analyses [36]. In this approximation the incident fields are averaged along the axis of the spheroid, and the average is used in the long-wavelength approximate equations developed for plane-wave analyses. The results obtained are surprisingly close to those calculated by more accurate methods. The long-wavelength approximation is very useful for near fields because the calculations are easy to make and the equations provide valuable insight.

The EBCM has also been used for near-field calculations [37]. The SARs produced in spheroids irradiated by short electric dipoles, small loops, and small-aperture fields have been calculated. Work is now under way to calculate the SAR in spheroids produced by the irradiation of larger aperture sources.

Calculations of the SAR in infinite cylindrical models of the human body have been made using techniques similar to those used for plane-wave irradiation. The resulting mathematical formulation was, of course, much more complicated.

Another technique used is to expand the incident near fields in terms of a spectrum of plane waves, then calculate the average SAR for each plane wave component by previously used techniques in block models of man [38]. Again, the calculations of the near-field SAR are much more complicated than those for plane-wave irradiation.

SOME RESULTS

Extensive SAR data are given in the Radiofrequency Radiation Dosimetry Handbooks [1, 35]. The second edition primarily contains SAR data for plane-wave irradiation; the third edition, the available data for near-field irradiation. The fourth edition, which is scheduled for release in 1985, will include material from previous editions along with more recent work. Since these extensive compilations of data are available, only examples of dosimetry results are given in this paper.

Figure 2, which shows the SAR and the function of frequency for plane-wave irradiation of an average-man model, illustrates how the SAR changes with polarization of the incident radiation. The strong resonance effect for the E polarization is also evident. A less pronounced resonance occurs with K polarization, and essentially no resonance with H polarization. The block model curve in Fig. 2 also shows smaller resonances that are produced by the other parts of the body, such as the head and arms. These minor resonances do not appear in the results calculated for spheroidal models.

Figure 5 shows a comparison between the SAR characteristics for an average man and a medium rat. The resonances, which are markedly different for the two models, occur at frequencies for which the length of the body is approximately four-tenths of a wavelength. At a given frequency, the internal field pattern in a man obviously differs greatly from that in a rat. More is said about this in the next section.

Ground-plane effects on the average SAR for man models is shown in Fig. 6. The main effect of the ground plane is to lower the resonant frequency. This occurs because the electric image of the man in the ground plane makes the length of the body appear to be twice as long as it would be in free space; thus the resonant frequency is approximately half that of the body in free space.

A comparison between calculated values for spheroidal models and some earlier experimental values in saline figurines is shown in Fig. 7. Another comparison between block-model calculations, VLF-MF measurements, and thermographic measurements is shown in Fig. 8. Other experimental data are summarized in the Radiofrequency Radiation Dosimetry Handbook [1, 35]. The experimental values in Fig. 7 are quite close to the measured values. The measured values in Fig. 8, though, are a factor of 2 or more higher than the calculated values for the block model. Hill [39] obtained similar results in measurements on human volunteers. His measured SAR values at frequencies in the 10-30-MHz range were about a factor of 2 greater than values calculated for spheroidal and block models. Hill proposed that the spheroidal models

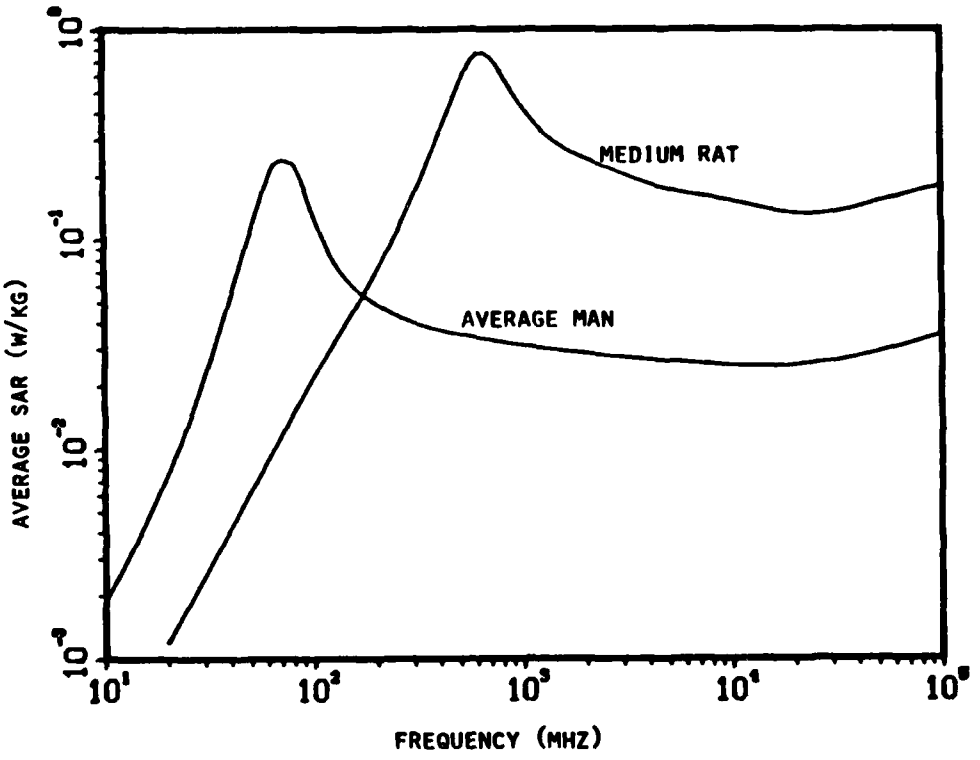


Fig. 5. Average SAR for prolate spheroidal models of an average man and a medium rat for E polarization, with an incident plane-wave power density of 1 mW/cm^2 [1].

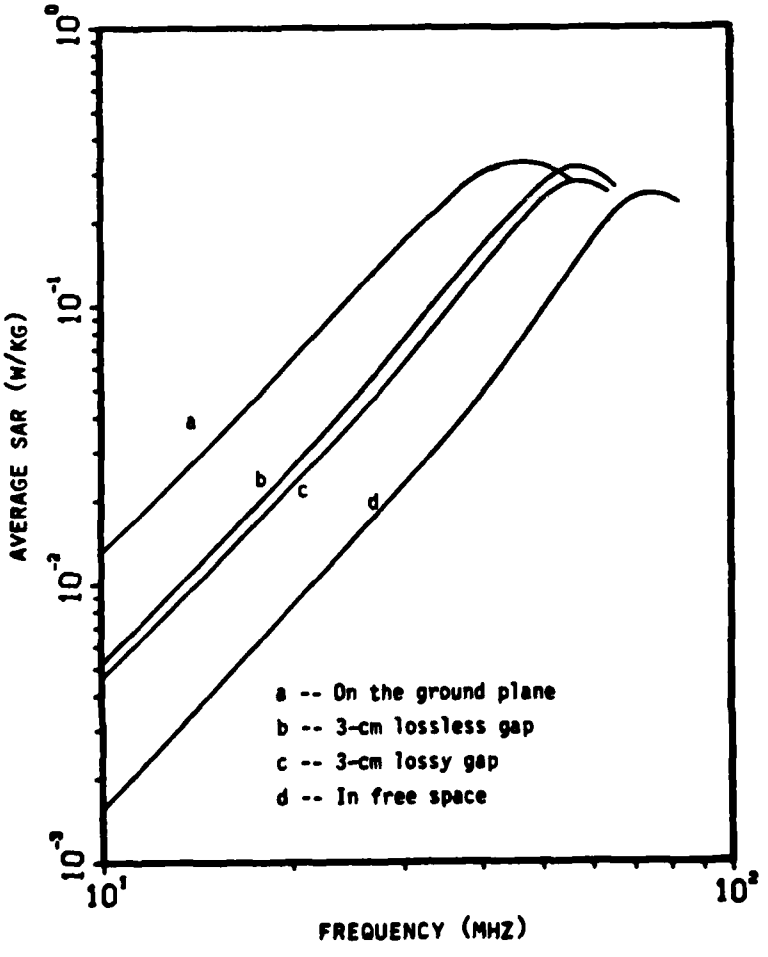


Fig. 6. Average SAR for an average man separated from a perfect ground plane by a gap of 3 cm, both with and without loss [1]. Curves for the man standing on the ground plane and in free space are shown for comparison.

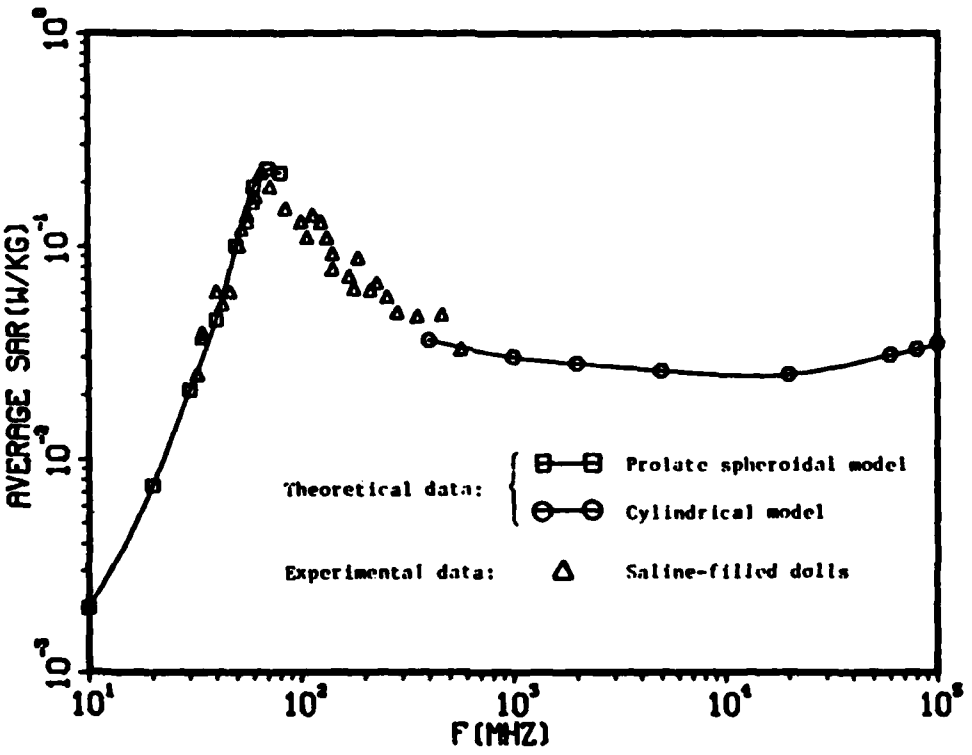


Fig. 7. Calculated and measured values of the average SAR for models of an average man, E polarization [1]. Incident power density is 1 mW/cm^2.

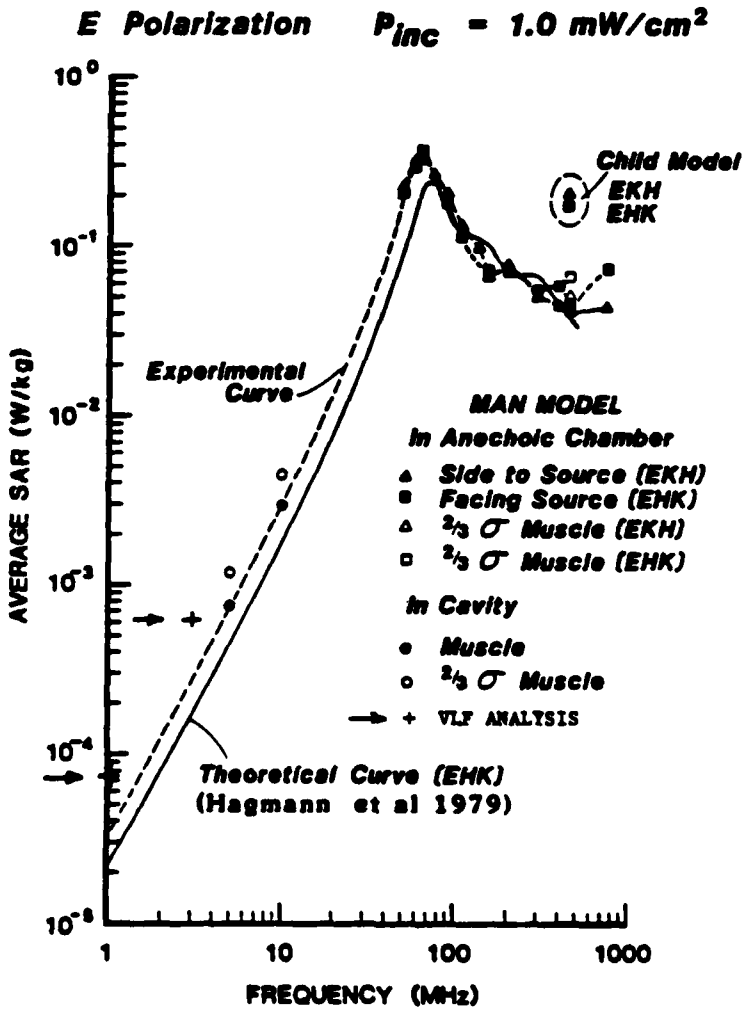


Fig. 8. Comparison of theoretical and experimentally measured whole-body average SAR for realistic man models exposed at various frequencies. The experimental curve is measured results--in scaled human-shaped models at simulated VLF frequencies--using thermographic techniques [32].

should be chosen on the basis of the ratio of the major axis to the minor axis rather than on the basis of major axis and mass, as usually has been done. He showed that thinner spheroids give results closer to measured values between 10 and 30 MHz.

As mentioned in the section Moment Method Using Smaller Cells, the calculations for SAR distribution using the block model and moment method with pulse basis functions have given inaccurate results, but calculations for average SAR using this method apparently gave good results. The experimental data in Fig. 8 and Hill's results seem to indicate that even the calculations for average SAR in the block model are not as good as supposed. This is reinforced by the fact that DeFord et al. [23] found the average SAR for the 1132-cell inhomogenous block model to be about twice that for the 180-cell homogeneous block model. Further refinements of the models will probably bring the calculated and measured values even closer together.

Figure 9 shows the average SAR produced in a spheroidal model of an average man irradiated by a

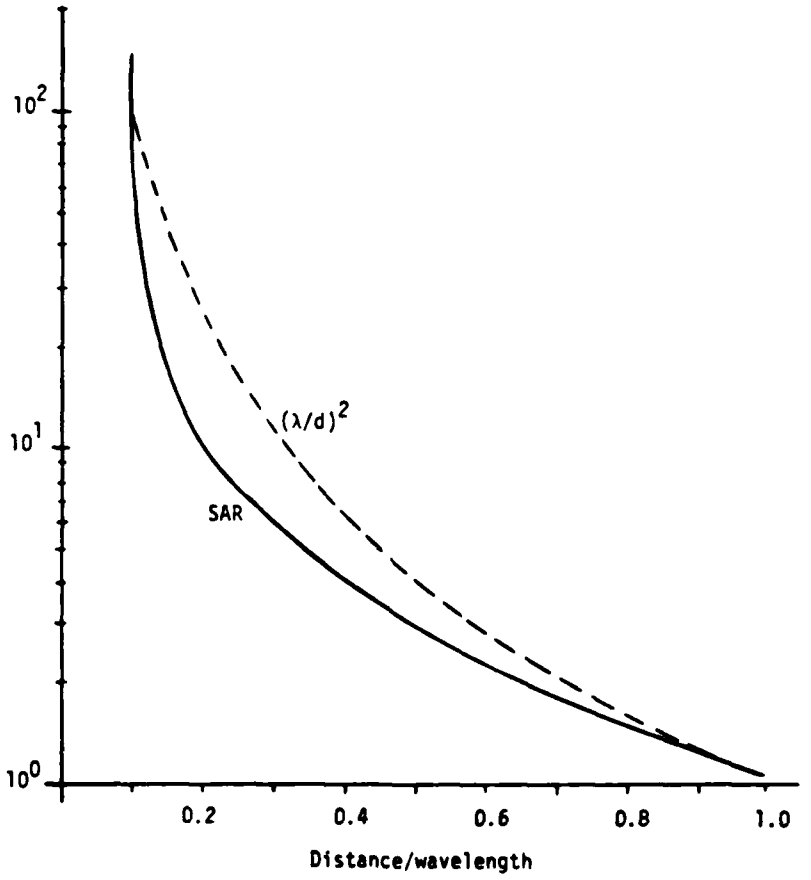


Fig. 9. Calculated values of normalized SAR and $(\lambda/d)^2$--for a spheroidal model of an average man irradiated by a short electric dipole--for E polarization at 200 MHz [1]. d = distance between the dipole and the spheroid; λ = wavelength of irradiation.

short electric dipole at 200 MHz as a function of the distance between the dipole and the spheroid. Note that the SAR does not increase as fast as the inverse distance squared--as might be expected from the 1/r variation of the far fields--which would correspond to a $1/r^2$ variation in the incident power density of the far fields. One might expect that in the near fields, the increase in the SAR would be greater than it would be in the far fields, since the near fields vary faster than 1/r. The reason that the SAR does not increase as fast as inverse distance squared is explained in the next section, in terms of the incident near-field variation. Similar behavior has been found in measurements of the average SAR in spheroidal models [40].

QUALITATIVE EXPLANATIONS OF ABSORPTION CHARACTERISTICS

Two qualitative principles can be used to predict relative average SAR values. Some examples of qualitative explanations based on these principles are given in this section.

Qualitative Principles

At the lower frequencies, the internal electric fields can be thought of as being generated by the incident $\underline{E}$ and $\underline{H}$ separately. That is,

$$\underline{E}_{in} = \underline{E}_{e} + \underline{E}_{h} \tag{3}$$

where

$\underline{E}_{e}$ is the internal electric field caused by $\underline{E}_{inc}$, the incident $\underline{E}$ field.

$\underline{E}_{h}$ is the internal electric field caused by $\underline{H}_{inc}$, the incident $\underline{H}$ field.

$\underline{E}_{in}$ is the total internal electric field in the body.

At the lower frequencies, $\underline{E}_{e}$ and $\underline{E}_{h}$ can be calculated separately from $\underline{E}_{inc}$ and $\underline{H}_{inc}$ and added to obtain $\underline{E}_{in}$, as given in equation (3). This is not true at higher frequencies, where $\underline{E}_{e}$ and $\underline{E}_{h}$ cannot be separately attributed to $\underline{E}_{inc}$ and $\underline{H}_{inc}$, respectively, since E and H are coupled together by Maxwell's equations. However, the general concepts based on equation (3) do seem to have some validity at higher frequencies, sometimes even up to resonance.

The following two principles can be used to predict the relative values of $\underline{E}_{in}$:

1. $\underline{E}_{e}$ is stronger when $\underline{E}_{inc}$ is more parallel to the boundaries of the object, and weaker when $\underline{E}_{inc}$ is more perpendicular to the boundaries of the object.
2. $\underline{E}_{h}$ is stronger when $\underline{H}_{inc}$ intercepts a larger cross section of the object and weaker when $\underline{H}_{inc}$ intercepts a smaller cross section of the object.

Figure 10 shows some examples of qualitative evaluations of internal fields based on these principles. For simplicity, only simple objects are used for illustration, but the principles apply to more complicated shapes like the human body.

Explanation of Polarization Effects

Figure 11 illustrates the qualitative explanation of the difference in average SAR shown in Fig. 2 for the three polarizations. For frequencies below resonance, the average SAR for E polarization is the greatest because both $\underline{E}_{e}$ and $\underline{E}_{h}$ are strong. The SAR for H polarization is lowest because both $\underline{E}_{e}$ and $\underline{E}_{h}$ are weak. The average for K polarization lies between that for E polarization and that for H polarization because $\underline{E}_{e}$ is weak, but $\underline{E}_{h}$ is strong. Thus the two qualitative principles very nicely explain why the SAR is a strong function of the polarization. Other similar qualitative explanations can be used to predict whether the average SAR will be large or small for a given set of conditions.

Explanation of Near-Field SARs

Similarly, the qualitative principles can be used to explain near-field absorption characteristics. Figure 12 shows the same information as Fig. 9, along with information about the electric field. Note that $|E|^2$ is less than $(\lambda/d)^2$, but follows along nearly parallel with it. The change in $|E|^2$ does not explain why the average SAR increases more slowly than $(\lambda/d)^2$ until it rises suddenly between λ/d = 0.2 and 0.3 and then rapidly increases above $(\lambda/d)^2$ for λ/d less than 0.2. The explanation is found in the change of the orientation of the $\underline{E}$ field with respect to the spheroid, as described by α, the angle between $\underline{E}$ and the major axis of the spheroid. Note that α is increasing from λ/d = 1 to about 0.25, where it suddenly begins to decrease. As α is increasing, E is changing from mostly parallel to more perpendicular to the spheroid. According to the first qualitative principle, this means that $\underline{E}_{e}$ is becoming weaker as α increases. Thus, $\underline{E}_{e}$ is getting weaker from about λ/d = 1 to about 0.25, and then becomes stronger as α decreases again. This factor causes the average SAR to begin to rise rapidly as α decreases. Thus we see that the qualitative principles can be used to explain near-field absorption characteristics as well as plane-wave absorption characteristics.

Adjustments for Frequency Differences

The dosimetric data shown in Fig. 5 allow researchers to extrapolate observed effects in animals to those expected in man. For example, it is clear from the curve that if a rat and a man were both irradiated by a 650 MHz plane wave, the average SAR in the rat would be more than an order of magnitude greater than that in the man. Hence, if a biological effect were observed in a rat with a given incident power density, a similar biological effect would be expected to occur in man at a much higher incident power density. Another important difference would be that the local SAR in the rat would be quite different from that in the man. In the man, the heating would tend to be more superficial than in the rat because the size of the man compared to a wavelength is much larger than the size of the rat.

An approximate numerical extrapolation of experimental results with animals to those that would be expected with man can be made on the basis of the kind of dosimetric information given in Fig. 5. For example, suppose that a biological effect, such as a behavior change, was observed in medium rats irradiated by EM plane waves of 5 mW/cm^2 incident power density at a frequency of 650 MHz, which is approximately the resonant frequency. For the local SARs to be similar in the rat and the man, the ratios of the wavelength to the body length should be approximately equal in each case. That is,

$$\frac{\lambda_r}{\ell_r} = \frac{\lambda_m}{\ell_m}$$

where λ_r and λ_m are the wavelengths for the irradiation of the rat and the man, respectively, and ℓ_r and ℓ_m are the lengths of the rat and the man, respectively. For ℓ_r = 0.2 m, ℓ_m = 1.75 m, and λ_r = 0.46 m (f

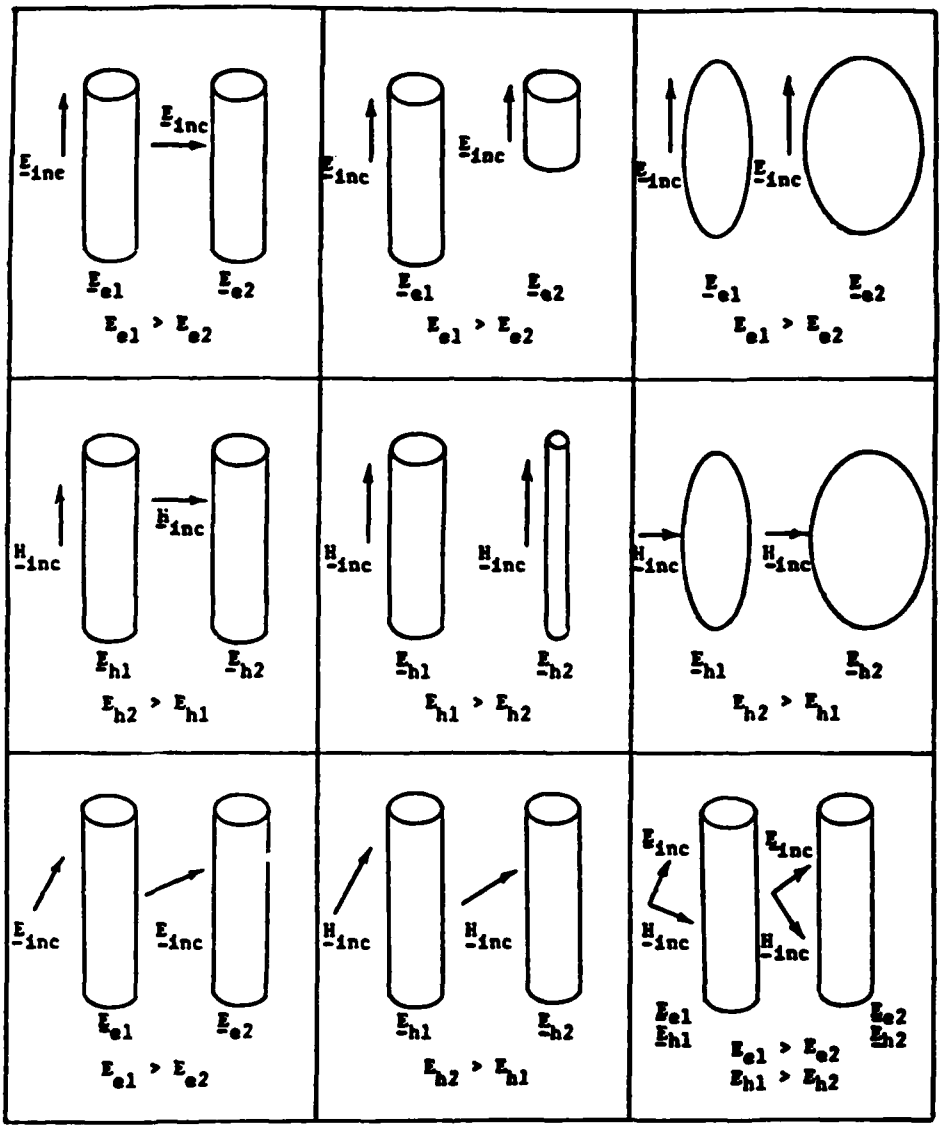
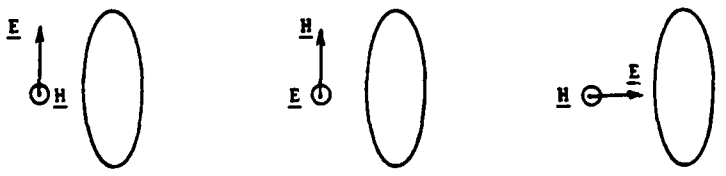


Fig. 10. Qualitative evaluation of the internal fields based on qualitative principles [35]. $\underline{E}_e$ is the internal electric field generated by $\underline{E}_{inc}$ (the incident E field), and $\underline{E}_h$ is the internal electric field generated by $\underline{H}_{inc}$ (the incident H field).



E Polarization	H Polarization	K Polarization
E_e strong	E_e weak	E_e weak
E_h strong	E_h weak	E_h strong

Fig. 11. Qualitative explanation of the differences in average SAR shown in Fig. 2 for the three polarizations in spheroidal models. ⊙ means the vector is normal to the paper.

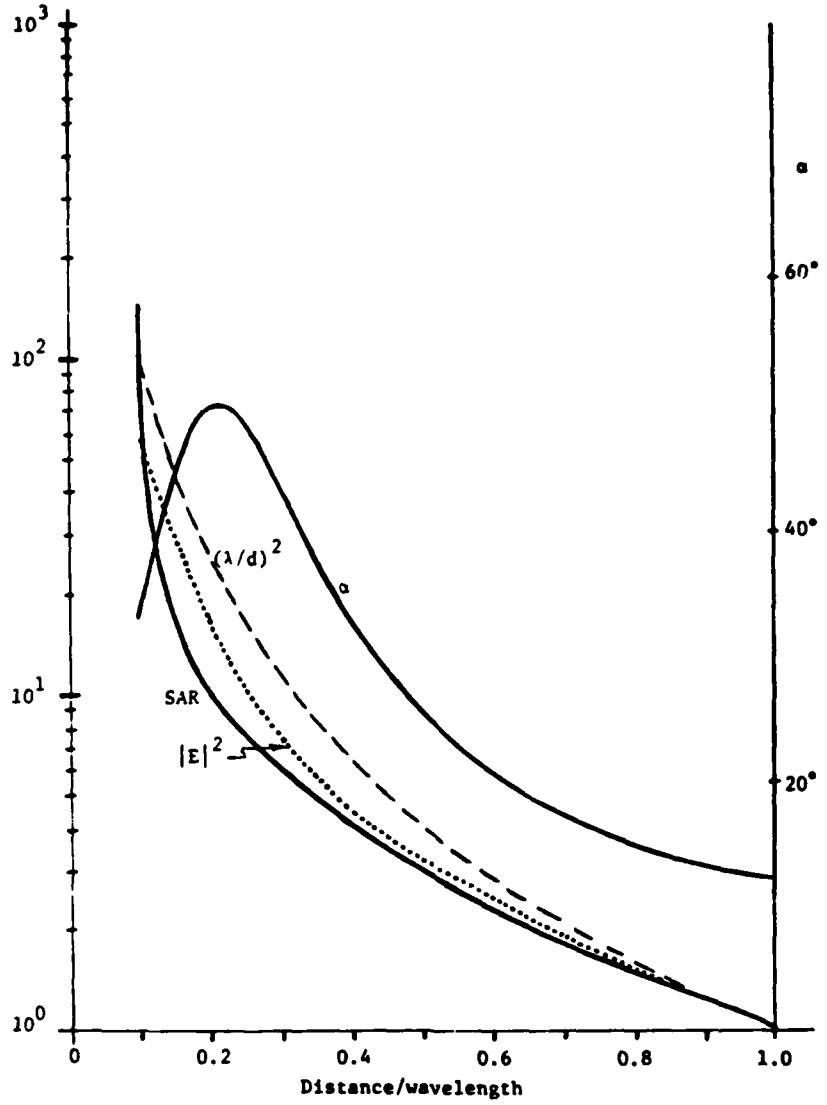


Fig. 12. Calculated values of normalized SAR, normalized $|E|^2$, and angle between $\underline{E}$ and the spheroid's major axis--for a spheroidal model of an average man irradiated by a short electric dipole for E polarization at 200 MHz. d is the distance between the dipole and the spheroid, λ is the wavelength of the radiation, and α is the angle between $\underline{E}$ and the spheroid's major axis.

= 650 MHz), the equation requires that λ_m = 40 m, which corresponds to a frequency of approximately 75 MHz. This makes sense because Fig. 5 shows that 75 MHz corresponds approximately to resonance in the man. Thus as far as local SAR distribution is concerned, a similar pattern would be observed in each case if the rat were irradiated at 650 MHz and the man at 75 MHz.

The incident power density at 75 MHz that would produce the same average SAR in man that occurred in the rat at 650 MHz can also be calculated from the curves in Fig. 5. At 650 MHz, the average SAR in the rat is about 0.8 W/kg for 1 mW/cm^2 and consequently is about 4.0 W/kg for 5 mW/cm^2. At 75 MHz, the average SAR in man is about 0.23 W/kg for 1 mW/cm^2. Thus an incident power density of 17.4 mW/cm^2 would be needed to produce 4.0 W/kg in man. So, if the change in behavior in the rat were related directly to the average SAR, a similar change in behavior in man might occur upon irradiation at 75 MHz with an incident power density of 17.4 mW/cm^2. However, a similar behavior change would probably not be expected to occur in man irradiated at 650 MHz with 5 mW/cm^2, because the average SAR would be much lower in the man than in the rat.

These examples show how important the calculated values of the SAR can be in interpreting experimental results. Without the two curves shown in Fig. 5, it would not be clear how observed experimental effects in animals might be related to expected effects in people. Theoretical dosimetry allows comparison, at least on the basis of average SAR and approximate local SAR distribution.

SUMMARY

What Can be Done

Plane Waves--Plane-wave dosimetry, both theoretical and experimental, is a reasonably well-developed discipline. Average SARs can be calculated for models of people and animals over a wide frequency range. For E polarization the SAR can be calculated from a simple empirical formula. Experimental measurements agree fairly well with the calculated results. Average SAR characteristics can be explained qualitatively.

Local SAR distributions can be calculated over a more limited frequency range, and above resonance (70 MHz in man), only at great expense. Local SAR distribution calculations have been verified by experimental measurements only to a limited extent. Usable models of man are not nearly as good for local SAR calculations as they are for average SAR calculations.

Near Fields--Near-field dosimetry is not as well developed as plane-wave dosimetry. Calculations have been made for SARs produced by simple sources, and calculations are being made for more complicated sources. Some experimental measurements of near-field SARs have been made. Average near-field SARs can be explained qualitatively, but there is no convenient normalization to incident power density for near fields as there is for plane waves. Near-field calculations are much more complicated than plane-wave calculations.

What Needs to Be Done

Plane Waves--More work in both theoretical calculation and experimental measurement of local SAR distributions needs to be done. Particularly, the local SAR distributions calculated by numerical methods need more verification, and more work should be done in calculating temperature distributions produced by RFR.

Near Fields--Theoretical calculations are needed for near-field SARs over a wider frequency range. Calculations for more realistic sources are also needed. Methods should be developed for calculating both local and average SARs produced by measured near fields. We need to be able to measure the incident fields at some specified points, and then calculate the expected SARs produced in people exposed to those fields. In addition, methods for calculating temperature distributions are needed.

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THERMAL CONSEQUENCES OF LOCALIZED SAR FROM RFR EXPOSURES

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Summary

Local SAR levels from 50 to 170 W/kg have been used for many decades for theraputic heating in clinical medicine. These levels can cause tissue temperatures to rapidly increase and stimulate a rapid increase in blood flow. Localized high levels of SAR can destroy tumors with minimal damage to healthy surrounding tissue due to the difference in blood cooling rates and thermal sensitivity of neoplastic as compared to normal tissues. SARs of 140 W/kg or greater can produce cataracts in the eyes of rabbits that are exposed locally to 150 mW/cm^2 cm 2450 MHz microwave radiation but similiar levels of exposure will not produce cataractogenic levels of SAR in primates. Experiments have shown that SAR levels of 72 W/kg or greater applied for 1.5 to 6 min to the brain of cats will produce temperature rises of 4.4 to 6.5 ^{o}C resulting in pulse rate increases, hyperventilation, bradycardia, and intense tachypnea for 15 to 20 minutes after exposure. No pathological changes were noted in the cat due to these localized exposure levels. Localized exposure of living cat brain at SAR levels of 2.5 to 5 W/kg can produce measureable temperature rises and changes in latency of evoked potentials. A most interesting thermal response occuring at the lowest known level of applied energy is that of microwave pulse hearing. The hearing response will be elicited with a temperature change of only 5 x 10^{-6} ^{o}C produced by an absorbed energy of 10 mJ/kg. On-the-other-hand, the SAR perception is 200 W/kg in the index finger where heat dissipation is high due to the large area to volume ratio. This is well above the threshold for tissue damage for the same SAR applied to other locations in the body.

List of Symbols

CT	critical temperature	d(ΔT)/dt	difference per unit time change in temperature
∇	gradient operator		
Δ T	temperature difference	g-cal/cm^2-sec	gram calories per square centimeter per second
E	electric field strength		
F	mass specific blood flow	k_2	0.698
RFPG	ANSI radio frequncy protection guide	kJ/kg	kilojoules per kilogram
RFR	radio frequency radiation	k_2	thermal conductivity of of tissue
SAR	specific absorption rate	kcal/kg-^{o}C	kilocalories per kilogram per degree celsius
T	temperature		
T_a	arterial temperature	kg/m^3	kilograms per cubic meter
T_o	initial temperature	mHz	megahertz
V/m	volts per meter	mJ/kg	millijoules per kilogram
W/kg	watts per kilogram	mW-cm-^{o}C	milliwatt centimeters degree celsius
W_a	specific absorption rate in tissue (W/kg)	mW/cm^2	milliwatts per square centimeter
W_b	heat loss due to blood perfusion (W/kg)	ml/100g-min	millliliters per 100 grams per minute
W_c	heat loss due to conduction (W/kg)	mm	millimeters
W_m	metabolic heating in tissue (W/kg)	^{o}C	degrees celsius
		^{o}C/sec	degrees celsius per second
W_o	initial metabolic heating rate	sec	seconds

c	specific heat	ρ	density of tissue
c_b	specific heat of blood	$_b$	density of blood
W	watt	mW	milliwatt
J	joule	mJ	millijoule
m	meter	cm	centimeter
min	minute	h	hour
sec	second		

1.0 INTRODUCTION

Thermally induced biological effects in human or animal subjects exposed to radiofrequency radiation (RFR) may result from either whole body systemic heating or partial body localized heating. The two modes of heating may have considerably different effects on the biological system. Generally the energy is absorbed very nonuniformly in the bodies of exposed subjects. The rate of absorbed energy which is a function of position and time may be expressed in terms of specific absorption rate (SAR) given in units of watts per kilograms (W/kg). The SAR at a particular point in the body may be considerably lower or considerably higher than the SAR as averaged over the whole body. For whole body exposures the peak or maximum SAR levels in both animals and man may be as high as 20 times the whole body average. A good example for human subjects is a case where a human standing on a conducting ground plane is exposed to plane waves at frequencies less than 30 MHz. For this case the SAR averaged over the ankles is approximately 20 times the whole body average. At other exposure frequencies between 450 and 900 MHz there may be selective absorption in various areas of the body leading to significantly higher local SAR than the whole body average. The highest levels of SAR usually occur in the fingers, the arms, or the legs depending on the exposure conditions. Higher localized SAR than whole body average may also occur from partial body exposures to clinical diathermy or cancer hyperthermia equipment, industrial radiofrequency heat sealers and small transmitting antennas associated with hand-held and mobile-communication units. According to the exclusion clause in the ANSI C95.1 - 1982 Radio Frequency Protection Guide (RFPG) (1), a whole body average SAR of 0.4 W/kg and peak localized SARs of 8 W/kg as averaged over any gram of tissue are allowed.

It is important to know the thermal consequences of high levels of local SAR. Local SAR levels from 50 to 250 W/kg have been used for many decades for therapeutic heating and clinical medicine. These levels can cause tissue temperatures to rise rapidly approaching 40 to 43°C which in turn can stimulate a rapid increase in mass specific blood flow from 10 to 40 ml/100g-min. This important technique, used in physical medicine and more recently in radiation oncology, will produce many physiological responses mostly due to direct thermal action on the tissue cells and the nervous system. Depending on the modulation characteristics of the RFR, the location of energy absorption, and the thermal regulation of the tissue where the energy is absorbed, the type of biological effects of RFR may vary over a wide range. Perception thresholds of energy absorption may vary from as low as a few mJ/kg for the auditory system to as high as 2 kJ/kg for the thermal receptors in the finger.

Scientific methods for measuring the RFR induced SAR and temperature distributions will not be discussed in this paper since they are discussed elsewhere. These methods include the use of thermography and phantom models reported by Guy (2), and Guy et al. (3); thermography on sacrificed animals reported by Johnson and Guy (4); RFR transparent temperature sensors (5); and the use of RFR transparent E field detectors (6). The following sections will discuss the thermal consequences of RFR on localized SAR in highly vascularized muscle or high water content tissue; poorly vascularized tissue such as subcutaneous fat, bone, and the eyes; central nervous system tissue; peripheral nerves; and the auditory system. The latter results from stimulation of the cochlea through vibrations produced by RFR induced thermoelastic expansion of tissues in the heads of exposed humans and animals due to nonuniform localized SAR.

2.0 THERMAL CONSIDERATIONS

In order to evaluate and understand the thermally induced biological effects of RFR exposure, one must know the relationship between the absorbed energy, the tissue cooling mechanisms, and the temperature. The energy equation for the time rate of change of temperature (°C/sec) per unit volume of subcutaneous tissue exposed to RFR is

$$d(\Delta T)/dt = 0.239 \times 10^{-3}[W_a + W_m - W_c - W_b]/c \qquad (1)$$

where W_a is the specific absorption rate (SAR), W_m is the metabolic heating rate, W_c is the heat loss due to thermal conduction, W_b is the power dissipated by blood flow, all expressed in W/kg, c is the thermal conductivity expressed in kcal/kg-°C, and $T = T - T_o$ is the difference between tissue temperature T and the initial temperature T_o. The SAR

for tissue exposed to an EM field is

$$W_a = \sigma E^2/\rho \tag{2}$$

where σ is the electrical conductivity in mhos/meter, ρ is the tissue density in kg/m^3 and E is the rms value of the electric field (V/m) in the tissue. Within the safe temperature range, the metabolic heating rate may be expressed as

$$W_m = W_o\ (1.1)^T \tag{3}$$

where W_o is the initial metabolic heating rate.

The thermal conduction term may be expressed as

$$W_c = (k_c/\rho)\ \nabla^2 T \tag{4}$$

where k_c is the thermal conductivity of the tissue in mW-cm-^oC and ∇ is the gradient operator.

If it is assumed that blood enters the tissue at arterial temperature T_a and leaves at tissue temperature T_b we may express blood cooling by

$$W_b = k_2 F c_b/\rho_b \Delta T\ , \tag{5}$$

where $\Delta T = T_a - T_b$, c is the specific heat of blood, ρ_b is the density of blood, F is the mass specific blood flow rate in ml/100g-m, and the constant $k_2 = 0.698$.

Prior to the time that RFR exposure is applied, it is assumed that a steady-state condition exists where $W_a = T = d(T)/dt = 0$, requiring

$$W_m - W_c - W_b = 0. \tag{6}$$

According to the typical values of the physical and thermal properties of tissues tabulated by Sekins and Emery (7), the equilibrium values of the terms in Eq. (3) under normal conditions are in the order of 1 W/kg for typical resting muscle. When a level of RFR capable of inducing an SAR of 20 - 200 W/kg is applied to tissue T, will increase, as shown in Figure 1, with an initial linear transient period typically lasting about 3 min for an SAR of 100 W/kg, where by Eq. (1)

$$d(\Delta T)/dt = 0.239 \times 10^{-3} W_a/c \tag{7}$$

This period, giving an initial time rate of temperature increase of approximately 1.73×10^{-2} ^oC/min per W/kg, is followed by a non-linear transient period usually lasting another 7 to 10 min, where T becomes sufficiently large that blood flow and thermal conditions become important in dissipating the applied energy. In tissues with negligible or insufficient blood flow, the temperature will monotonically approach a steady-state value dictated by the magnitude of W_a, as shown on the upper curve where equilibrium is reached when $W_a + W_b = W_c$. For vascularized tissue, however, blood flow plays a significant part in heat dissipation, limiting the slope of the d(T)/dt curve. In addition, for vascularized tissues, a marked increase in blood flow will occur due to vasodilation when the tissue is heated to a critical temperature (CT) in the range 42 to 44 ^oC. As a result, the temperature will rapidly change and approach a steady-state value at a somewhat lower level, as shown in the figure, when $W_a = W_c + W_b - W_m$, indicating a significant reserve of blood cooling capacity. For proper and safe therapeutic action when using RFR for diathermy purposes, it is necessary to raise the temperature sufficiently in the deeper vascularized tissue to trigger the vasodilation without exceeding safe levels in the poorly vascularized intervening subcutaneous fat layer. Clinical experience has shown that when normal vascularized tissue is exposed to a diathermy source, pain will be felt by the patient before any tissue damage can occur. In fact, the pain may be used as a guide to indicate that the tissue temperature has reached the required 43 to 45 ^oC for vasodilatation and associated therapeutic benefits. When the RFR is used for treating cancer, sufficient SAR must be used to raise the tumor temperature to destructive levels without harming the normal tissue. The blood flow cooling rate in the normal tissues will generally be higher, since the tumor blood flow will not increase as sharply as the blood flow for normal tissue under RFR exposure.

The SAR, W_a, must be sufficiently high so that the therapeutic level of temperature can be maintained over the major portion of the treatment period. If too little power is applied, the period of elevated temperature will be too short for any benefits. If too high a level is applied, the temperature can overshoot the safe level before the vasodilatation can take effect. The pain sensors are a reliable and sensitive means for detecting this temperature range, however, and if the applied power level is set so that mild pain or discomfort is first felt by the patient, the vasodilatation will be sufficient to limit the temperature to even a lower level that is both tolerable and

therapeutically effective. If the effective temperature is reached at the surface, it is felt as a mild burning sensation. On the other hand, if it is reaches the deeper tissues, it is felt as a dull-aching type of pain. Hardy (8) has shown that the intensity of absorbed non-penetrating radiation at the threshold of pain in the skin is 0.045 g-cal/cm^2-sec, or 188 mW/cm^2. The thermal consequences of localized exposure of various types of tissue are discussed below.

ABSORPTION IN MULTILAYERED TISSUES OF HIGH AND LOW WATER CONTENT

The thermal consequences of localized SAR in layered living tissues have been studied for many years in relation to therapeutic application of RFR. Especially important has been the problem of quantifying these effects in both deep, high water content tissue and the surface of subcutaneous tissue during clinical diathermy applications. Schwan (9, 10, 11) theoretically demonstrated the dependence of relative heating in the tissue on the thickness of the skin, the thickness of the subcutaneous fat, and the frequency of RFR (plane wave) normally incident on the surface of the skin. Johnson and Guy (4) used Schwan's equations to obtain the results shown in Figures 2 and 3. The results show typical SAR characteristics from plane wave irradiation of the tissue for various diathermy frequencies (433 MHz authorized only for European use). Figure 2 illustrates the results for a wave transmitted through a subcutaneous fat medium into a muscle medium. The absorption is normalized to unity in the muscle at the fat-muscle interface. The relative absorption curves in the fat will remain the same for smaller fat thicknesses (e.g., the portion of the curves between -2 and 0 cm would correspond to a 2 cm-thick fat layer). Figure 3 illustrates the SAR in the muscle interface and in a 2 mm-thick skin layer as a function of fat thickness for an incident power intensity of 1 mW/cm^2. The values may be used to determine the SAR at other locations in the muscle and fat by relating them to the curves in Figure 2. The peak SAR density is always maximum in the skin layer for this type of tissue geometry. The curves illustrate that 1) absorption is so great at 2450 MHz in the muscle layer that the depth of penetration is only 1.7 cm, 2) the severe discontinuity at the fat-muscle interface produces a large standing wave resulting in a "hot spot" in the fat layer one-quarter wavelength from the muscle surface, and 3) the SAR in the deep tissues varies considerably with fat thickness. The curves indicate, however, that penetration into the muscle increases and subcutaneous fat absorption decreases at lower frequencies. Though there are no data in the literature on the SAR measured in actual human tissues exposed to plane wave RFR, a considerable number of measurements have been made in relation to clinical research on therapeutic applications of radio frequency (RF) fields from small applicators.

Of significance in the use of RFR for diathermy treatments or for selective heating of tumors as an adjunct to ionizing radiation treatment of cancer is the dependence of the blood-perfusion field upon changing tissue temperatures, and the RFR induced energy deposition or SAR pattern. The range between therapeutic and toxic temperatures can be narrow and the effectiveness of the therapy requires that the temperature elevation be spatially limited (local treatment). Guy et al. (12) and Lehmann et al. (13) quantitatively estimated the peak mass blood flow rates occurring after CT onset in RFR diathermy treatments; values ranging from 13.5 to 33 ml/100g-min. More recently Sekins (14) and Sekins and Emery (7) conducted an experimental and numerical investigation of the temperature and blood-flow fields during localized exposure of the human thigh to a 915-MHz diathermy applicator as shown in Figure 4. The applicator developed by Guy and associates (15), is designed to directly couple radiofrequency (RF) energy to the tissue with minimal stray radiation losses, allowing accurate measurement and control of the RF energy dose. The applicator utilized the flow of cold air through a plastic surface grid at its aperture to prevent unacceptably high skin and subcutaneous tissue temperatures, providing a means shifting or "focusing" the thermal energy deeper into the muscle. Figure 5 illustrates how the "depth focused" energy deposition is obtained by superimposing the effects of surface cooling (curve 2) with those of RF deep heating (curve 3), yielding a temperature profile whose temperature peak is shifted further into the muscle bed (curve 4) away from the fat-muscle interface.

Using experimental and theoretical techniques, Sekins developed a model for predicting the relationship between mass blood flow temperature and SAR. The parameters for blood-flow rate were determined experimentally for various temperatures by using xenon clearance rates after injection of the tracer in a particular volume of tissue. By combining this information with the theoretical thermodynamic equations, Sekins was able to compare experimentally measured temperature versus time curves with theoretical predictions as shown in Figure 6 for a typical subject subjected to localized RF energy deposition in the thigh. Sekins used a finite element analysis to evaluate the theoretical values. The spatial distribution of experimental and theoretically determined temperature versus time are shown in Figure 7 for the same subject. The shading denotes the region of the subcutaneous fat. Finally, the numerically determined steady state temperature, SAR and BFR profiles for a number of subjects are shown in Figure 8. Sekins (14) data show that the BFR and SAR curves are similar in shape but the maximum levels of the former are shifted deeper into the tissue relative to the maximum level of the latter and the temperature curves. Thus, the proportionality between local blood flow rate and local temperature is not the sole basis of the physiological response to heating. The results show that when the local steady state tissue temperature is below an injurious level and the BF response has adjusted to the relative magnitudes of heat loss via blood flow and conduction to create an energy balance, the blood flow can be significantly depressed in the region of maximum temperature. Under these conditions the vascularized muscle tissue tolerates SAR levels

exceeding 120-220 W/kg without pain or injury.

The increase in blood flow due to vasodilitation is accompanied by increases in capillary pressure, cellular membrane permeability, and metabolic rate. It is believed the responses can increase healing rate in diseased or damaged tissue by increasing the transfer of metabolites across cell membranes, providing for greater concentration of white cells and antibodies, and increasing transport rate of toxins, engulfed bacteria, and debris away from the related area. The heating can promote relaxation of muscles, reduce pain, and provide relief of muscle spasms. Localized high levels of SAR can destroy tumors with minimal damage to healthy surrounding tissue due to the difference in blood cooling rates and thermal sensitivity of neoplastic as compared to normal tissues.

RFR-INDUCED CATARACTS IN RABBITS

In the above section it was demonstrated that healthy vascularized muscle tissue could tolerate 120-220 W/kg without pain or thermal damage. However, for poorly vascularized areas such as the lens of the eye, the threshold for tissue damage is generally less than that for other tissues.

Production of lens opacification in the eyes of laboratory animals by exposure to microwave radiation has been known to occur since 1948 (16-18). However, the exact conditions under which these changes exhibit themselves were not fully quantified until recently (19-26).

Absorption of microwave energy in the eye and consequent conversion into heat has been the most accepted mechanism responsible for the cataractogenic effect. Some reports (24-25), however, suggest that some factors other than the thermal one might be responsible. These reports allude to formation of opacities or other changes in the lenses or corneas of animals receiving single or repeated exposures of microwave radiation at levels believed to produce insufficient temperature rise to produce thermal damage. A large portion of early investigations were characterized by lack of quantitative rigor and produced few results useful for the purpose of scientific extrapolation to human exposures. More recently, quantitative relationships between the physical variables of microwave radiation and the biological changes in the eye have been determined so that animal data can be used for predicting safe levels of human exposure.

By quantitative measurement of the actual fields or SAR in the affected tissue structure relative to the incident radiation, the microwave field and power patterns both inside and outside the rabbit's head and eyes have been established. The measurements were made while the organ was exposed to near zone 2450-MHz radiation from a corner reflector (diathermy C director). Time and power threshold for cataractogenesis in rabbits exposed to the above conditions were determined. In vivo experiments and computer modeling have been applied to study the intraocular temperature during microwave irradiation. The good agreement between these two approaches reinforces the suggestion of a thermal mechanism for micowave cataractogenesis.

MEASUREMENT OF SAR IN EXPOSED EYES OF RABBITS

In a series of experiments where rabbit eyes were locally exposed to 2450 mHz RFR, a comparison was made between power density, SAR, temperature and threshold for cataractogenisis (19-23). In one group of animals the SAR distribution along the anterior-posterior axis of the eye and extending to the head was determined by measuring the temperature rise in the tissue, which is proportional to the absorbed energy, due to a short exposure to high-intensity radiation. The SAR was then calculated from equation (7).

A fast-reacting, iron-constantan thermocouple was passed into a probe inserted into the eye and the intraocular temperature was measured at increasing depths of 2-mm increments, each taken just prior to and just after a short exposure (20 sec) of a known level (540 mW/cm^2) of high-intensity microwave radiation. The measurements in the rabbit head extending beyond those of the eye were conducted at 3-mm intervals immediately after sacrifice. An overdose of anesthetic solution was used to sacrifice the animals.

The SAR patterns resulting from the dosimetry measurements are shown in Figure 9. In all cases, the absorption reached peak values within the vitreous body, about 1.5 cm behind the cornea, and had a mean of 0.92 W/kg per mW/cm^2 incident power density. During the dosimetry runs the rabbits genrally showed an increase in pulse rate of approximately 30 percent.

MEASUREMENT OF TEMPERATURE IN EYES OF EXPOSED RABBITS

In connection with the above measurements, another group of anesthetized animals were exposed to determine parameters associated with thresholds of cataractogenesis. A probe similar to that employed in the dosimetry studies was inserted 4 mm behind the superior limbus. The glass probe was oriented under direct visualization to lie just behind the posterior pole of the lens. Orbital, rectal, and ambient temperatures, as

well as respiratory rates and ambient percent humidity, were monitored throughout. Microwave irradiation at a predetermined level was performed at 5-min intervals, pausing momentarily for introduction of the thermocouple into the probe. After a 2-3 sec stabilization, the temperature was recorded and the thermocouple withdrawn. The process was repeated until the desired length of exposure was realized.

The actual peak temperature in the vitreous body behind the lens reached in three different radiation conditions was determined and is shown in Figure 10. These are for 100 mW/cm^2 incident for 60 min, 200 mW/cm^2 for 35-40 min, and 300 mW/cm^2 for 30-35 min. The SAR, as determined from Figure 9 is, respectively, 90, 180, and 270 W/kg. Each power level reached its own specific temperature plateau after 15-20 min due to the gradual increase of body-core temperature. The temperature for the 100 mW/cm^2 ones stayed consistently at 41 °C, while that for 200 and 300 mW/cm^2 all surpassed 41 °C. The baseline orbital temperature in each case was practically identical with the rectal or core temperature (Figure 11), but after the first 5 min of irradiation, the former followed the intravitreous temperature rise more closely. The orbital temperature, however, never rose as high as the vitreous. This presumably is because of the greater blood flow and hence greater heat-regulating capacity of the orbit. It is interesting to note that rabbits exposed to 200 and 300 mW/cm^2, without surgical manipulation of the eye or ocular temperature probing, all developed lens opacities while those exposed to 100 mW/cm^2 remained unaffected.

CATARACTOGENIC TIME AND POWER THRESHOLD FOR AN ACUTE SINGLE EXPOSURE IN RABBITS

The investigators conducting the above experiments also used another group of eighty-one rabbits, averaging 4.0 kg, to determine the cataractogenic threshold of near-zone 2450-MHz radiation. The rabbits' eyes were examined with the slit lamp immediately after irradiation and periodically thereafter. Any abnormalities were recorded using a camera attached to the slit lamp and by hand sketches. Depending upon the level of exposure, varying degrees of injection, tearing, pupillary constriction, and anterior-chamber turbidity were noted immediately following the exposure. These changes were transient and disappeared by the second day following exposure. Changes in the lens were usually detectable on the first or second day postirradiation. At the lower exposure levels, mild and often reversible changes were observed. These consisted of a milky band (single, double, or triple) in the posterior cortex, close to the posterior capsule and extending to the equator. This banding was visible only with the slit lamp. In addition, a chain of vacuoles or small vesicles formed in the area of the posterior suture. At high levels of irradiation, more advanced and permanent cataractous changes were noted. These changes consisted of more pronounced banding, an incease in the number of vacuoles with a definite well-circumscribed opacity forming in the posterior cortex, which was easily seen with an opthalmoscope. occasionally, large vesicles were seen in the equator of the lens, and, in a few cases, the posterior cortical opacity was found to involve not only the equator, but also extended from the equator to the anterior subscapsular cortex. As a rule, however, the cataracts were confined to the posterior cortical area. Only at relatively high levels would the entire lens become involved in the cataractous process. Examination of the fundi showed no abnormalities. The left, or control eye, aside from a transient pupillary constriction immediately following exposure, remained normal.

The time and power threshold resulting from these experiments is shown in Figure 12, along with Carpenter's (24) and Williams' (27) curves. Each point on the charge represents 3-6 animals. It can be seen readily that the threshold curve follows Carpenter's very closely. The maximum absorbed power shown in the right-hand side is slightly lower than the incident power density in numerical value. Note that the minimum cataractogenic power density was found at 150 mW/cm^2 for 100 min, which represents a maximum SAR power of 138 W/kg.

The temperature distribution of an intact eye in the rabbit under micowave irradiation was modeled numerically using a finite element of rectangular and triangular annular regions shown in Figure 13. The model incorporates into it various cooling mechanisms such as conduction, evaporation, and radiation (20). Calculations for 2450-MHz induced temperature distribution in the eyes of rabbits were performed for incident radiation levels of 100, 150, 200, and 300 mW/cm^2, applied for 100, 100, 30, and 20 min, respectively. The resulting banded isotherms over an anterior-posterior section are shown for several cases in Figures 14-17. In all cases, the highest temperatures are localized on the center line near the posterior surface of the lens. The 100 mW/cm^2 case showed slight elevation over the 39 °C blood temperature, whereas the 150, 200, and 300 mW/cm^2 produced temperatures of 40.0, 42.4, 42.8, and 45.8 °C respectively. These results were in complete agreement with the measured retrolental temperature rise.

RFR CATARACTOGENESIS IN PRIMATES

Research by Kramar et al. (23) has shown that the threshold for cataractogenesis in the primate is much greater than that for rabbits, probably due to the greater shielding protection of the eye by the orbital tissue surrounding it. In this research the SAR patterns were determined in three adult female rhesus monkeys. Intramuscular ketamine was used for premedication, and intravenous pentobarbital was injected via a cannulated femoral vein during surgical manipulations of the eye during irradiation. The monkey was restrained in a specially constructed lucite seat in a semi-reclining position. The

back of the scalp was shaved and the head glued into position in a concave pillow of styrofoam. The experimental set-up was similar to that of rabbits in the first two animals, except that a resonant slot radiator centered 5 cm over the bridge of the nose was used instead of the dipole cornea reflector combination.

In a third monkey, the slot was centered over one eye rather than over the nose. The distance between the slot and cornea was 5 cm. As shown in the results, this second position of the slot was necessitated by excessive heating of the nasal bridge which resulted from the centrally oriented applicator.

A total of four rhesus monkeys (two males, two females) were used in an attempt to find the cataractogenic threshold. One female monkey was irradiated at the incident power density of 300 mW/cm^2 for twenty-two minutes. During this exposure, the slot was centered over the nose. The remaining three monkeys were exposed with the slot centered over the right eye. The incident power levels used were as follows: 400 mW/cm^2 for sixty minutes, and 500 mW/cm^2 for sixty minutes.

With the slot centered over the nose, the greatest deposition in the eye was found in the vicinity of the lens (Figure 18). The peak absorbed power here was 2.9 W/kg per watt input to the applicator. However, with this orientation of the slot, the skin of the nose was damaged to the point where the nasal bone became exposed during the experiment. The power absorption pattern in one monkey with the slot applicator in this second position showed the peak SAR in the lens to be 3.5 W/kg per watt input to the applicator (Figure 19). This was significantly higher than the SAR of 2.9 W/kg per watt input to the applicator found in the lens when the slot was centered over the nose (Figure 18). Although this asymmetric orientation increased the heat deposited in the eye, the actual peak SAR was not in the lens but in the anterior chamber (Figure 19). One rhesus monkey (female) was irradiated with the slot centered over the nose, at an incident APD of 300 mW/cm^2 for twenty-two minutes. Second to third-degree burns were noted on the bridge of the nose, but the eyes remained unaffected. Varying degrees of nasal burns also resulted in the three monkeys irradiated with the slot centered over the right eye, at incident APD of 400 mW/cm^2 for thirty and sixty minutes, and 500 mW/cm^2 for sixty minutes. In addition, the highest incident APD power level (500 mW/cm^2 for sixty minutes) was followed by lid edema, contracted pupil and a moderately severe reaction in the anterior chamber. The anterior chamber reaction persisted for ten days. The lenses, however, remained clear in all three animals for a period of thirteen months.

NUMERICAL RESULTS FOR SAR AND TEMPERATURE IN EXPOSED MONKEY EYES

Because of the configuration of the facial bones, the deposition pattern in the monkey is much different from that of the rabbit. As a consequence, the local peaking of temperature at the posterior surface of the lens is not observed. Figure 20 is a typical isotherm pattern for the monkey. A smooth temperature profile exists throughout the eye with a very minimal rise in temperature. The peak retrolental temperature following a radiation level of 300 mW/cm^2 was only 40.2 ^{o}C in contrast to 45.1 ^{o}C found in the rabbit. Even following radiation levels of 500 mW/cm^2, the maximum retrolental temperature reached would be approximately 42 ^{o}C (Figure 21).

The thermal model (Figure 21) predicted that even at incident APD of 500 mW/cm^2 cm the critical ocular temperature in the monkey was barely 42 ^{o}C and hence no lens damage was to be expected without severe facial burns. This in turn was substantiated by the actual irradiation of monkeys. Rabbits, however, developed cataracts at significantly lower incident power density (Figure 12). The computer thermal model showed that in this species the critical ocular temperatures were well above cataractogenic threshold levels (Figure 22). The most apparent explanations for the species difference are anatomical configuration and dimensions. Whereas the rabbit has very prominent eyes and little surrounding skeletal protection, the monkey's eyes are deep-set and shielded by well-developed brows and orbital rims. In man one would expect the degree of ocular prominence and hence vulnerability to show great individual variation. An additional factor might be the relative as well as the absolute size of the lens. For example, the antero-posterior diameter of the adult rabbit lens is approximately 7 mm, which represents almost one-half of the entire globe's diameter. In the rhesus monkey and in man the lens is about 4-mm thick and occupies one sixth of the antero-posterior diameter of the eye. Perhaps these smaller lenses can be cooled more effectively by the surrounding fluid.

The good agreement between the measured and the computed temperature fields found in these experimental animals suggests that it is possible to predict the ocular temperatures and hence the cataractogenic thresholds for man, if the blood flows and the SAR patterns are known. The SAR patterns can be obtained from model studies. However, there is currently insufficient data about the blood-flow rate to permit realistic estimates of thermal cataractogenic thresholds to be made.

LOCALIZED EXPOSURE OF CNS SYSTEM

Another area of the body more vulnerable to RFR induced energy absorption is the central nervous system. Lin et al (28) conducted a series of experiments to determine the tolerance of the brain to localized RFR heating in relation to possible use of the energy for producing differential hyperthermia as an adjunct for combination therapy for

brain cancer. The authors exposed the heads of cats to RFR levels of 95, 190, and 380 mW/cm^2 for periods of 360, 180, and 90 seconds respectively. SAR distribution measured by Johnson et al (4) on phantom, dead, and living cat brains exposed to the applicator are given in Figure 23. The data in Table 1 is obtained by combining these results with temperature measurements by Lin et al (28). Because the histologic effect of microwave irradiation on the brain tissue is of particular interest and because difficulties of measuring the brain temperature in vivo, without introducing damage to the brain tissue, the brain temperatures indicated in Table 1 were measured using one set of animals exposed under the same irradiation conditions as another set of animals where physiological and histological effects were observed. While Lin et al (28) were able to raise the brain temperature drastically, the rectal temperature remained relatively normal. Although the dosages of microwave irradiation were the same for all cases, the temperature differential was smaller for the two low intensity, long duration exposures. Figure 24 indicates that this was due to the "radiator" effect of the cerebral vascular system and averaging effect of the whole body. The blood supply of the brain is greater than that of any other tissue in the body. In all of the animals the pulse rates increased simultaneously with microwave irradiation, and bradycardia occurred immediately after a period of hyperventilation. This was followed by an intense tachypnea. The rapid panting ended about 15-20 minutes after irradiation. The animals were maintained for 6 days, and on the seventh were sacrificed. No gross tissue changes were observed in the brains of any of the animals studied. Several of the brains were studied histologically with immediate fixation methods, but no microscopic changes were seen. However, one cat which had received 380 mW/cm^2 for 90 seconds displayed some degree of motor difficulty, indicating that neurophysiologic dysfunction had developed. This may have been due to the microwave or anesthesia since the animal remained unconscious for over eight hours. However, physiologic changes in nervous tissue may not parallel histologic changes and the finding of histologically normal tissue does not rule out changes in nerve functions produced by microwaves.

Guy et al (29) measured effects of localized exposure on the somatosensory and auditory systems of cats exposed by the same apparatus used in the above experiment. Electrophysiological responses due to electrical stimulation of the tactile receptors of the cat's contralateral forepaw, recorded from the somatosensory thalamus area of the cat brain with and without the presence of 918 MHz RFR were determined. The measureable effects of the microwaves appears to be an induced temperature rise in the thalamus with an associated decrease in latency time of neural responses within the exposed area. Figure 25 shows the evoked thalamic nucleus activity in an area (medial geniculate nucleus) involved in auditory mechanisms, and Figure 26 shows the same type of activity recorded for the same area due to stimulation of the skin on the contralateral forepaw during intermittent exposure to RFR. The peak microwave power absorption density and temperature in the thalamus area are noted on each curve. The latency times between the stimulus and the initial thalamus response (denoted by the first arrow) and between the initial thalamus response and a distinguishable later event (denoted by the second arrow) are also noted. The latency between the stimulus artifact and the first arrow represents the conduction time of sound in air and the propagation time of neural impulses to the recording area, whereas, the latency between the arrows is probably more associated with pathways within the brain. Resting periods between the 15 min exposure were increased for the higher power exposure in order to allow the brain tissue to cool sufficiently. Temperature changes and heart rate as shown in Figure 27 for the auditory experiments and in Figure 28 for the tactile system studies. Note the increase in heart rate for the higher exposure power levels. Experimental results given in Figure 29 indicate that both latencies decrease with increasing body temperature of the cat when produced by a hot pad, whereas, microwaves applied to the head have more of an effect on the later latency. The changes are reversible and have time constants that seem to be directly associated with the thermal effect of microwaves. The threshold for both temperature changes and latency changes was found to corrrespond to a maximum power absorption level between 2.5 and 5 W/kg at the center of the brain. It would take a plane wave power density of 10 to 25 mW/cm^2, however, to produce the same power absorption in the human brain assuming it can be represented by a simple sphere. Figure 30 presents the results from exposing the animal to the same total energy at different power levels and exposure times corresponding to a total incident energy of 1.0 $mW\text{-}h/cm^2$. This induces a peak specific absorption density of 0.78 W-h/kg as calculated from thermocouple measurements. This incident energy level corresponds to the former ANSI C95.1-1974 standard for minimum energy for human exposure for 1.0 $mW\text{-}h/cm^2$ or less. The same energy was applied with increasing incident power for decreasing exposure duration. The only effect is the slight characteristic change in latency as observed with the cw exposures mentioned previously. The maximum incident power used was 1 W/cm^2 for 3.5 sec, producing a peak SAR of 950 W/kg . Figure 31 illustrates the thalamic temperature changes associated with the exposures. Note that exposures of constant energy for 3 min or less result in approximately the same temperature increase of 0.2 to 0.3 ^{o}C.

Elevation of thalamic temperature by the circulation of heated fluid through a heat exchanger applied to the base of the skull resulted in evoked potential changes comparable to those produced by microwave heating of the same magnitude. In contrast to an earlier observation that heating of the whole cat by means of a heating pad yielded decreased latency in both early and late components of the evoked potential, the exchanger-heated cats showed changes only in the late components. This is directly analogous to the microwave case. This demonstration supports the contention that the above observed microwave effect is a thermal phenomenon. Figure 32 represents even more convincing support for this view. This figure shows the sequential pattern of brain temperature and evoked potential latency with successive application of radiation alone,

cooling alone, radiation combined with cooling, and during periods in which the animal's own temperature compensation mechanisms are in operation (i.e., during "recovery" from a radiation elevation, or a coolant depression of temperature). It is very evident that the evoked potential changes are associated with particular direction and magnitude of temperature change. With appropriate titration of coolant temperature against radiation energy, it is even possible to reverse the change that would be anticipated with radiation alone.

The above work supports the contention that evoked potential changes seen with CW microwave irradiation of the CNS are limited to those that can be attributed to thermal loading.

There appears to be no remarkable differences, at least within the resolving power of the evoked potential method, between tactile and auditory stimuli with equivalent CW radiation parameters. Brief pulse radiation, on the other hand, has produced, in classic auditory elements, highly specific effects. Figure 33 shows activity in the medial geniculate aea of the thalamus evoked by conventional acoustic stimuli (a "click" derived from a pulsed loudspeaker, a "click" provided by piezoelectric crystal cemented on the skull) and similar activity evoked by application of microwave pulses in both the UHF and microwave bands. The effect was also found at various frequencies between the two bands. Though the details of these experiments have been published elsewhere (30-35) the results will be summarized here. The threshold of the 2450 MHz microwave pulse evoked response as a function of pulse width is shown in Table 2 for a cat and Table 3 for humans. The thresholds for the evoked responses with microwave pulses 0.5 to 10 usec in duration appear to be related to the incident energy density per pulse at a level about one-half of that which produced audible sensations for the human exposure. The required threshold energy per pulse seems to increase with pulse width for 10 to 32 usec duration pulses with the exception of the 25 usec case. The peak absorbed energy density per pulse in the head of the cat was obtained from thermographic measurements. The incident energy density per pulse corresponding to the threshold for evoked responses recorded from the medial geniculate body due to 918 MHz radiation, differed very little from that for 2450 MHz. Most research results on this phenomenon indicate that the auditory effect is a result of rapid thermal expansion of tissues in the head resulting from an RFR induced inhomogeneous temperature distribution. The expansion sets up a brief vibration of the head at a natural resonant frequency inversely proportional to the diameter of the head (approximately 7 - 10 kHz for adult humans, 31 - 40 kHz for cats and 60 - 70 kHz for rats). The minimum temperature increase for producing this phenomenon has been calculated to be as low a 5×10^{-6} °C.

SAR AND ENERGY THRESHOLD FOR PRODUCING THERMAL PERCEPTION IN HUMAN FINGER

In general most RFR exposures result in maximal SAR in tissue structures that are long and small in cross-section such as the neck, upper and lower limbs, and the fingers for human subjects, and the legs and tails for laboratory animals. Fortunately these regions also receive maximal cooling due to thermal conduction and blood flow since the ratio of surface area to volume is large. Thus they are able to tolerate relatively high values of SAR. To demonstrate this the author conducted experiments to determine perception thresholds for RF current flow through the index finger. Table 4 illustrates the energy rrequired to produce thermal perception at the middle phalanx of the author's index finger, in the region of smallest cross section when an RF current was passed through the finger . A wet saline sponge electrode was placed in contact with the distal phalanx of the finger and a ground electrode was placed around the arm. Applications of 150 kHz current at levels of approximately 50, 80, and 100 mA were used and the time to perception was noted. Application times of 2 to 4 seconds for the 100 mA current were required to produce perception in both the right and left fingers corresponding to a total absorbed energy density of 837 to 2087 J/kg. The times to perception increased to approximately 9 seconds for 80 mA current (approximately 2500 J/kg) and to 32-44 seconds for the 50 mA current (approximately 3800 to 5300 J/kg). The SAR corresponded to 410-505 W/kg, 257 W/kg and 90 - 121 W/kg for the three respective current levels used. The average conductivity and dielectric constant of the middle phalanx were obtained from impedance measurements between the proximal and distal joints of the finger with results as shown at the bottom of Table 4.

Additional experiments were done on human volunteers of various weight and sizes to determine threshold of perception of RF currents 50 Hz to 150 kHz. Some typical results are shown in Table 5 for both a heavy female and a very light female. Below 90 kHz the sensation was electric shock, but above 90 kHz the sensation was thermal. The current was stepped using a double ascending staircase described by Cornsweet (36). The results in the table are averages of 5 samples with each series lasting approximately 3 to 4 minutes. Since the average of the staircase current during the period of each series of tests was slightly below the perception level, the values given in the table can be assumed to be slightly above steady state levels of perception current. Note that the maximum SAR for the 2.0 cm^2 cross section finger was 123 W/kg while the value for the smaller 1.5 cm^2 cross section finger was as high at 1370 W/kg indicating the greater heat dissipation properties of the smaller finger. These results indicate that the 8 W/kg peak SAR for the ANSI C95-1982 standard may be too conservative for exposure of the fingers and limbs of the body.

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TABLE 1. Brain and Body Temperature Changes Due to Selective Brain Heating [Lin et al, (28)]

Exposure condition			Temp. Increase °C	
Exp. Time (s)	Power Density (mW/cm^2)	SAR (W/kg)	Brain	Rectal
90	380	153	6.5	0
180	190	77	4.8	0.1
360	95	38	4.4	0.1

Table 2. Threshold of Microwave Evoked Auditory Responses in Human (2450 MHz 3 pulses/sec, Background Noise 45 db)

Peak Power Den. (W/cm^2)	Average Power Den. (uW/cm^2)	Pulse Width (us)	Energy Dens. /Pulse (uJ/cm^2)	Absorbed Energy/Pulse[1] (mJ/kg)
40	120	1	40	16
20	120	2	40	16
13.3	120	3	40	16
10	120	4	40	16
8	120	5	40	16
4	120	10	40	16
2.33	105	15	35	14
2.15	129	20	43	16
1.8	135	25	45	18
1.25	120	32	40	16

1. Based on absorption in equivalent spherical model of head.

Table 3. Threshold of Microwave Evoked Auditory Responses in Cat (2450 MHz 3 pulses/sec, Background Noise 45 db)

Peak Power Den. (W/cm^2)	Average Power Den. (uW/cm^2)	Pulse Width (us)	Energy Dens. /Pulse (uJ/cm^2)	Absorbed Energy/Pulse[1] (mJ/kg)
35.6	17.8	0.5	17.8	10.1
17.8	17.8	1	17.8	10.1
10.0	20.3	2	20.3	11.6
5.0	20.3	4	20.3	11.6
4.0	20.3	5	20.3	11.6
2.2	21.6	10	21.6	12.3
1.9	28.0	15	28.0	15.9
1.7	33.0	20	33.0	18.8
0.6	15.2	25	15.2	8.7
1.5	47.0	32	47.0	26.7

TABLE 5. PERCEPTION CURRENT, CURRENT DENSITY, AND SAR AS A FUNCTION OF FREQUENCY MIDDLE PHALANX OF RIGHT INDEX FINGER

87.7 kg Female Subject

AREA (cm^2) 1 FNG ,2 HAND, 3 WRST, 4 ANKLE= 2.0 4.5 23.0 43.6

FREQ kHz	I(MAN) mA	I(CMP) mA	SDEV mA	J(MAN) mA/cm^2	J(CMP) mA/cm^2	SAR W/kg
0.05	0.38	0.32	0.01	1.92E-01	1.64E-01	4.28E-03
0.10	0.41	0.33	0.01	2.07E-01	1.68E-01	4.50E-03
0.22	0.57	0.48	0.01	2.87E-01	2.44E-01	9.50E-03
0.50	0.54	0.52	0.02	2.72E-01	2.63E-01	1.11E-02
1.00	0.95	0.78	0.01	4.82E-01	3.95E-01	2.50E-02
2.00	1.27	1.12	0.03	6.41E-01	5.65E-01	5.11E-02
5.00	2.72	2.23	0.08	1.37E+00	1.12E+00	2.02E-01
8.00	4.05	3.31	0.11	2.04E+00	1.67E+00	4.46E-01
10.00	5.33	4.63	0.19	2.69E+00	2.33E+00	8.72E-01
20.00	11.07	9.05	0.27	5.59E+00	4.57E+00	3.34E+00
30.00	17.48	14.32	0.57	8.82E+00	7.23E+00	8.36E+00
40.00	24.30	20.35	0.14	1.23E+01	1.03E+01	1.69E+01
50.00	30.65	26.52	0.34	1.55E+01	1.34E+01	2.87E+01
60.00	36.89	32.91	1.35	1.86E+01	1.66E+01	4.42E+01
70.00	39.66	33.56	1.41	2.00E+01	1.69E+01	4.59E+01
80.00	58.18	46.75	1.56	2.94E+01	2.36E+01	8.91E+01
90.00	63.57	49.64	3.73	3.21E+01	2.51E+01	1.00E+02
100.00	67.71	55.00	1.74	3.42E+01	2.78E+01	1.23E+02
150.00	64.39	52.87	3.21	3.25E+01	2.67E+01	1.14E+02

47.7 kg Female Subject

AREA (cm^2) 1 FNG ,2 HAND, 3 WRST, 4 ANKLE= 1.5 133.6 15.8 37.7

FREQ kHz	I(MAN) mA	I(CMP) mA	SDEV mA	J(MAN) mA/cm^2	J(CMP) mA/cm^2	SAR W/kg
0.05	0.26	0.27	0.02	1.75E-01	1.86E-01	5.52E-03
0.10	0.34	0.32	0.01	2.30E-01	2.15E-01	7.42E-03
0.22	0.51	0.44	0.03	3.45E-01	2.97E-01	1.41E-02
0.50	0.57	0.60	0.02	3.87E-01	4.08E-01	2.67E-02
1.00	1.11	1.06	0.02	7.57E-01	7.25E-01	8.41E-02
2.00	1.71	1.68	0.02	1.17E+00	1.14E+00	2.09E-01
5.00	3.15	3.28	0.07	2.15E+00	2.24E+00	8.01E-01
8.00	5.07	5.55	0.07	3.46E+00	3.78E+00	2.29E+00
10.00	6.27	7.51	0.10	4.28E+00	5.12E+00	4.19E+00
20.00	16.73	18.68	0.19	1.14E+01	1.27E+01	2.60E+01
30.00	24.49	31.42	0.48	1.67E+01	2.14E+01	7.34E+01
40.00	40.85	42.06	0.36	2.79E+01	2.87E+01	1.32E+02
50.00	42.93	50.25	0.53	2.93E+01	3.43E+01	1.88E+02
60.00	68.51	67.34	3.25	4.67E+01	4.59E+01	3.37E+02
70.00	94.04	98.74	2.95	6.41E+01	6.73E+01	7.25E+02
80.00	98.06	119.92	2.98	6.69E+01	8.18E+01	1.07E+03
90.00	110.49	130.26	2.89	7.53E+01	8.88E+01	1.26E+03
100.00	118.58	135.53	4.18	8.09E+01	9.24E+01	1.37E+03
150.00	158.23	131.26	4.02	1.08E+02	8.95E+01	1.28E+03

Table 4. Energy Corresponding to Thermal Perception of 150 kHz Current Through Middle Phalanx of Index Finger

	CURRENT (mA)	TIME (s)	CURRENT DEN. (mA/cm2)	SAR (W/kg)	ENERGY (J)
RIGHT FINGER	100.0	2.63	50.7	410.2	1078.8
	100.0	2.04	50.7	410.2	836.8
	100.0	2.27	50.7	410.2	931.1
	107.0	3.00	54.3	469.6	1408.9
	108.0	2.19	54.8	478.4	1047.8
	107.0	2.13	54.3	469.6	1000.3
	107.0	2.87	54.3	469.6	1347.8
	108.0	2.67	54.8	478.4	1277.4
	111.0	4.13	56.3	505.4	2087.2
	110.0	3.63	55.8	496.3	1801.6
	Mean = 1282 SD = 399.4 J				
	80.7	9.46	40.9	267.1	2527.1
	80.5	8.41	40.8	265.8	2235.5
	Mean = 2381 SD = 206.2 J				
	54.4	43.80	27.6	121.4	5316.8
	53.9	32.20	27.3	119.2	3837.2
	Mean = 4577 SD = 1046. J				
LEFT FINGER	109.0	2.20	48.1	371.1	816.5
	109.0	3.25	48.1	371.1	1206.2
	110.0	3.49	48.6	378.0	1319.1
	111.0	3.07	49.0	384.9	1181.6
	Mean = 1131 SD = 218.0 J				
	80.3	9.63	35.5	201.4	1939.7
	80.2	9.89	35.4	200.9	1987.1
	Mean = 1963 SD = 33.5 J				
	54.0	19.47	23.8	91.1	1773.5

Cross Sectional Area of Right Finger = 2.26 square cm
Cross Sectional Area of Left Finger = 1.97 square cm
Measured Impedance Proximal to Distal Joint = 214.2 - j15.3 ohms
Measured Impedance Proximal to Distal Joint = 215.1 - j18.0 ohms
Calculated Conductivity of Right Finger = 0.627 S/m
Calculated Conductivity of Left Finger = 0.624 S/m
Calculated Dielectric Constant of Right Finger = 5372
Calculated Dielectric Constant of Left Finger = 6275

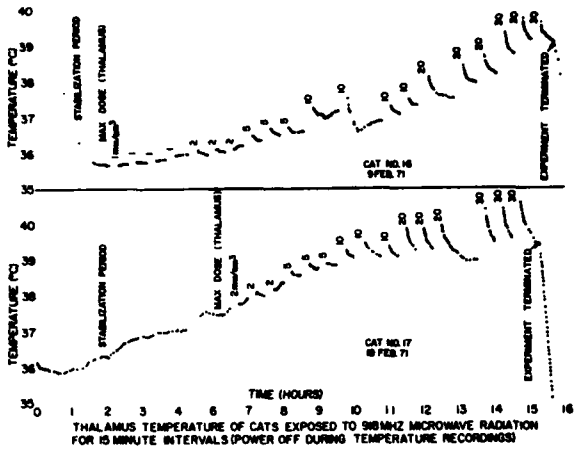


Fig. 28. Thalamic temperature of cats exposed to 918 MHz microwave radiation for 15 min intervals (power off during temperature recordings) (4)

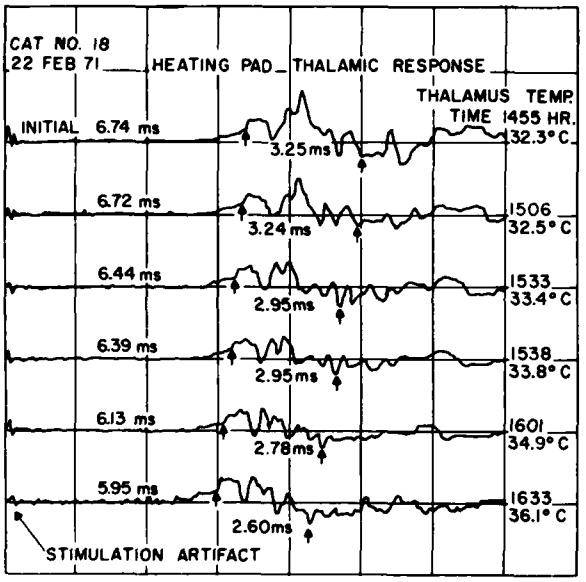


Fig. 29. Heating pad effects on thalamic response of cat to stimulation of contralateral forepaw (29)

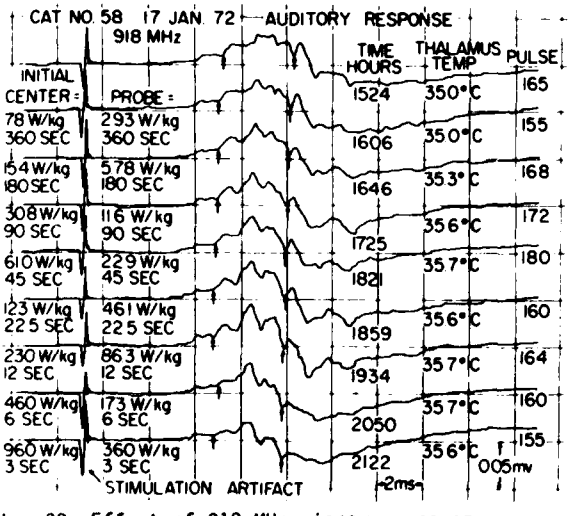


Fig. 30. Effect of 918 MHz microwave power radiation on the evoked auditory response of medial geniculate body to an acoustic click stimulus (29)

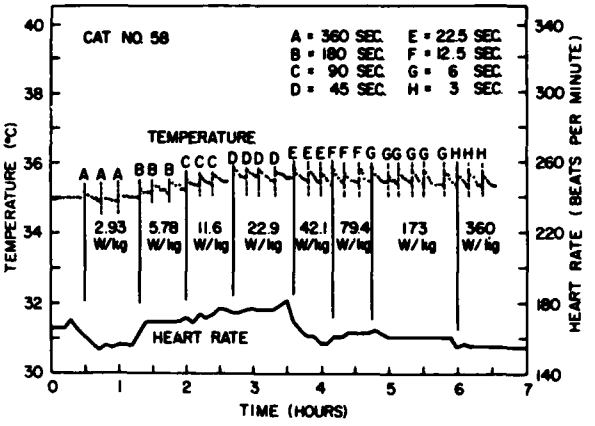


Fig. 31. Thalamic temperature and heart rate of cat exposed to 918 MHz microwave radiation (29)

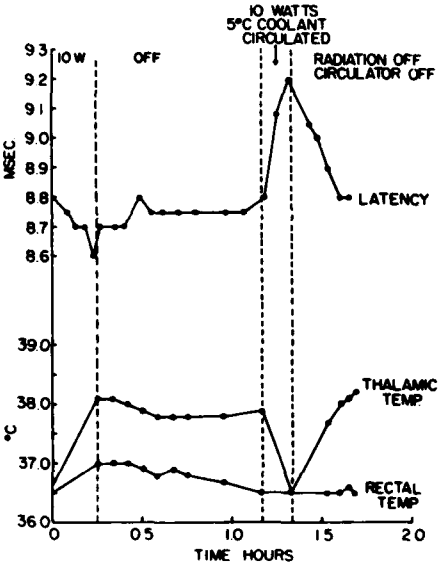


Fig. 32. Sequential 918 MHz CW microwave radiation

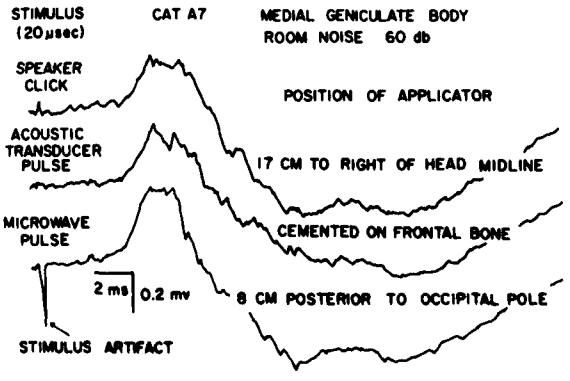
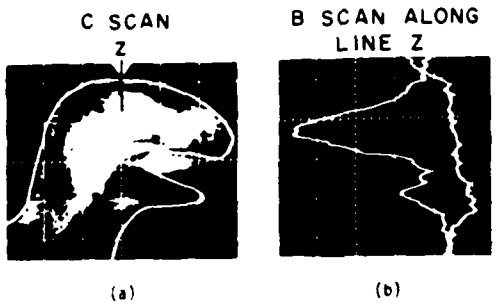


Fig. 33. Evoked responses recorded from medial geniculate body of the cat (29)



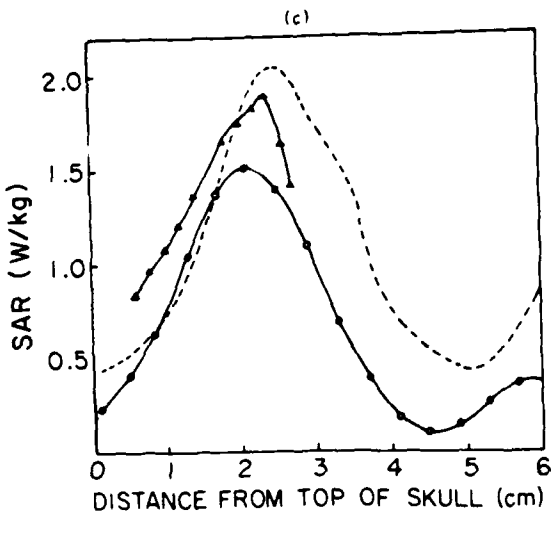


Fig. 23. SAR in cat's head exposed to 918 MHz radiation (28)

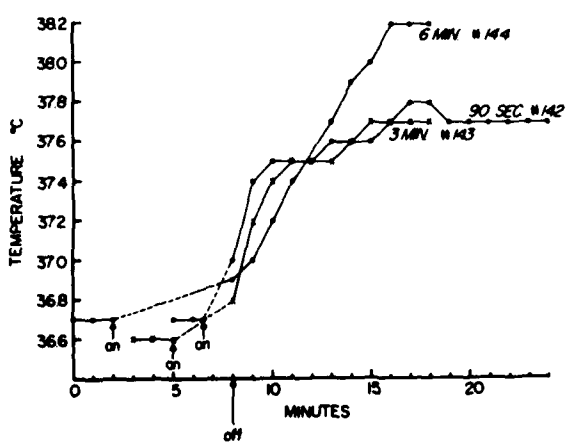


Fig. 24. Body temperature during selective microwave brain heating (918 MHz) (28)

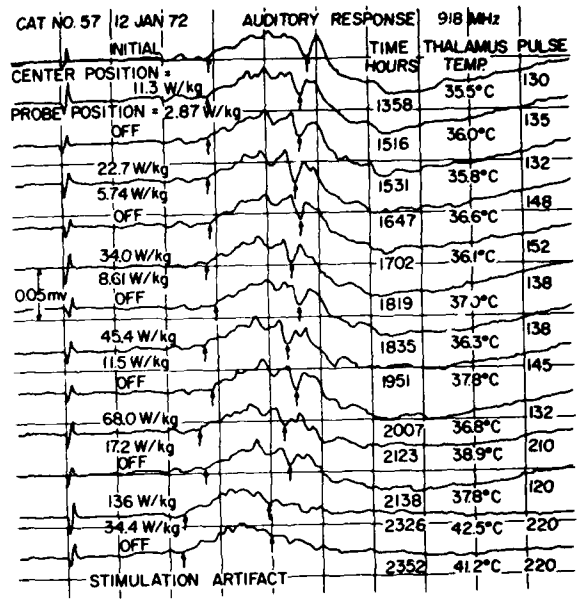


Fig. 25. Effect of 918 MHz microwave power radiation on the evoked auditory response of the medial geniculate body to audio click stimulus (29)

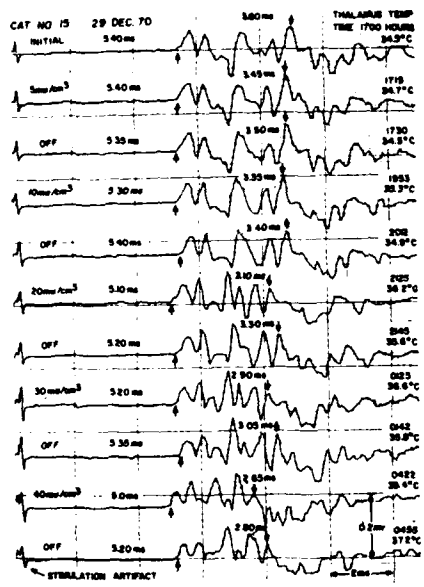


Fig. 26. Effects of microwave radiation on thalamic response of cat to stimulation of contralateral forepaw (4)

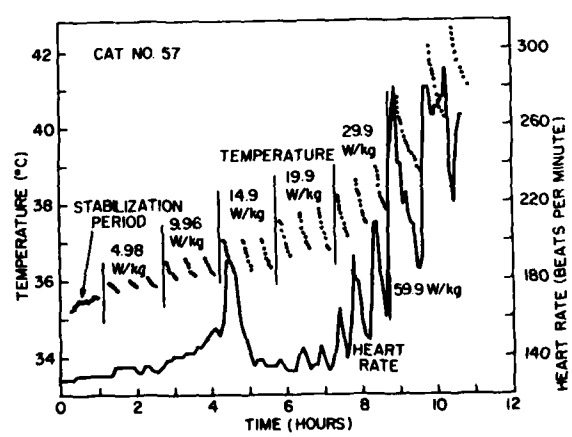


Fig. 27. Thalamic temperature and heart rate of cat exposed to 918 MHz microwave radiation (29)

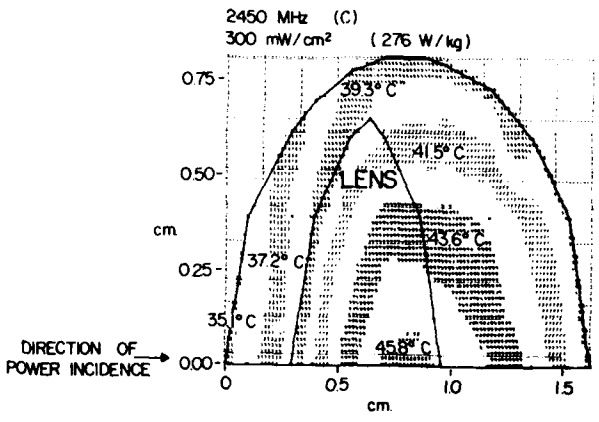


Fig. 17. Computer-predicted intraocular temperature in the rabbit exposed for 20 min at 300 mW/cm² (26)

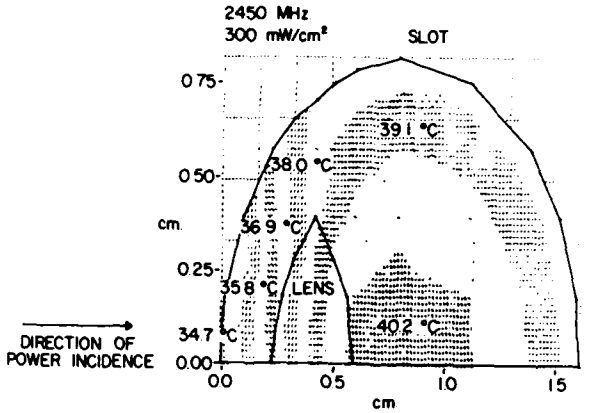


Fig. 20. Computed intraocular temperature in monkey eye exposed for 200 min at 300 mW/cm² (23)

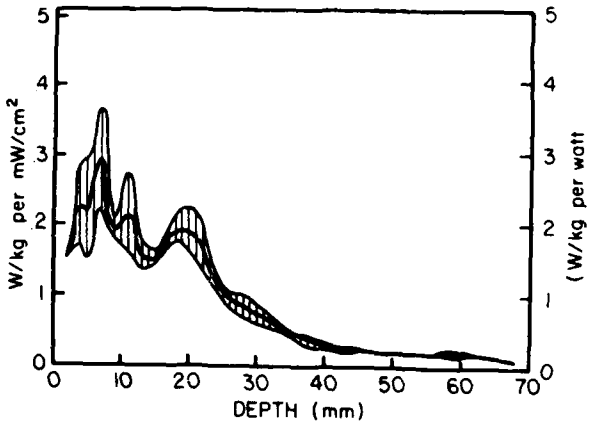


Fig. 18. SAR in eye and head of monkeys exposed to resonant slot source (23)

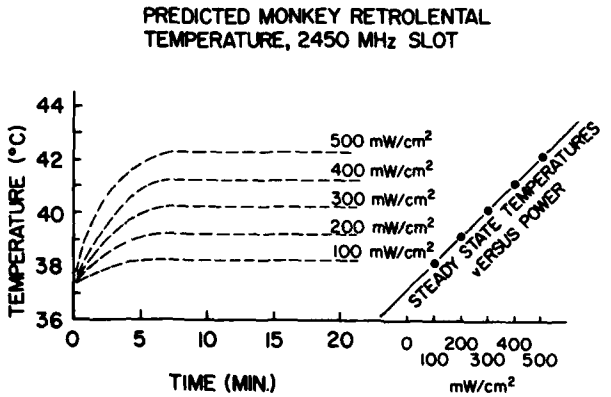


Fig. 21. Predicted peak retrolental temperatures in monkeys (23)

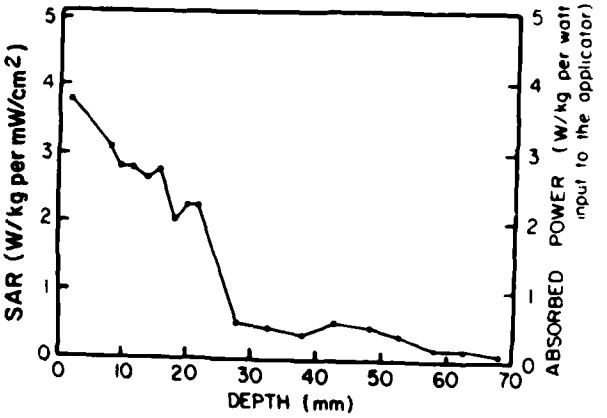


Fig. 19. SAR in eye and head of monkey exposed to slot source centered over right eye (23)

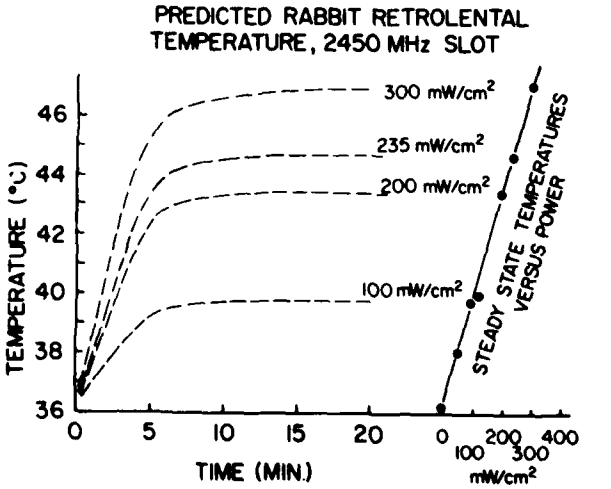


Fig. 22. Predicted peak retrolental temperatures in rabbits (23)

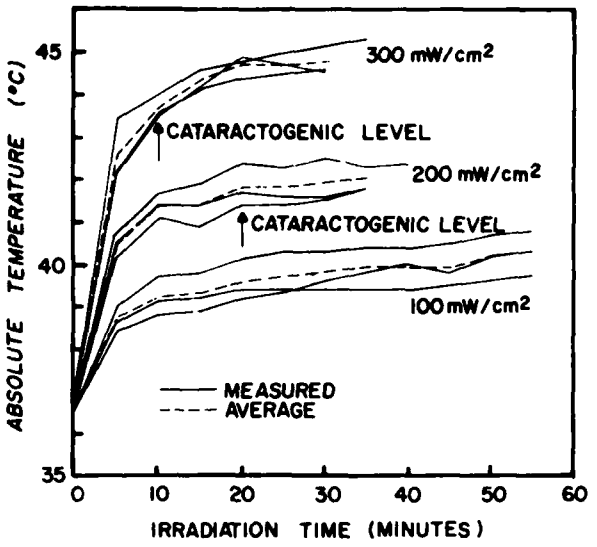


Fig. 10. Measured retrolental temperature in rabbits exposed to near-zone 2450 MHz microwaves (26)

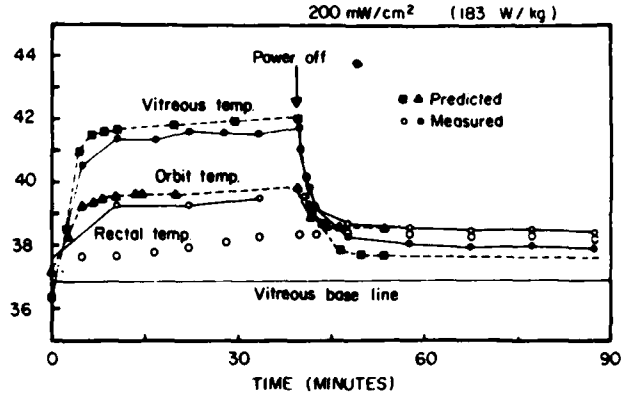


Fig. 11. Measured and predicted temperature changes in rabbits exposed to microwave radiation (26)

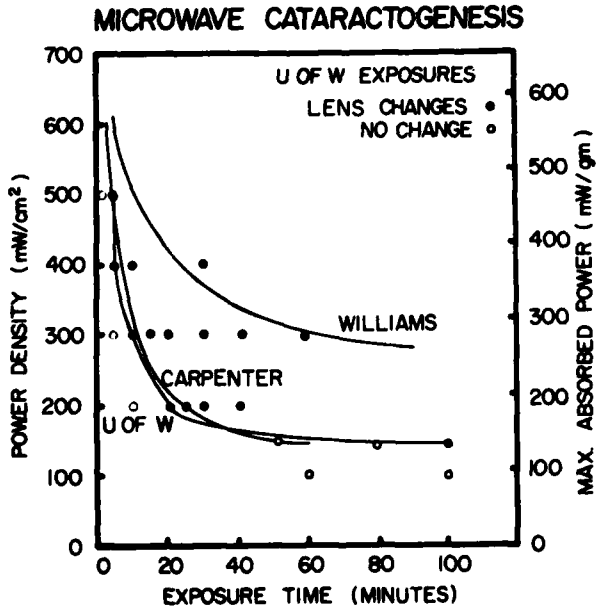


Fig. 12. Threshold for cataractogenesis in rabbits exposed to near-zone 2450 MHz radiation (26)

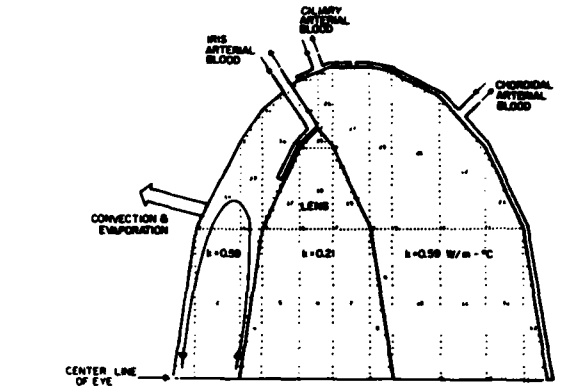


Fig. 13. Computer model of the upper half anterior-posterior section of rabbit eye (26)

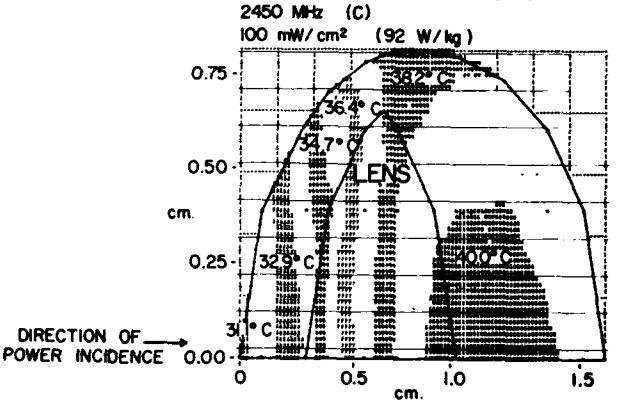


Fig. 14. Computer-predicted intraocular temperature in the rabbit exposed for 100 min at 100 mW/cm² (26)

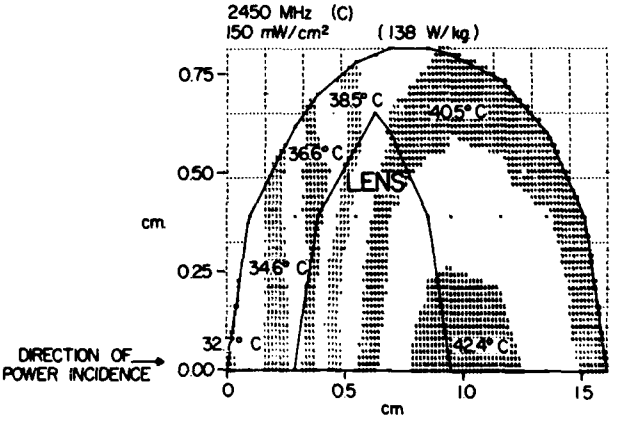


Fig. 15. Computer-predicted intraocular temperature in the rabbit exposed for 100 min at 150 mW/cm² (26)

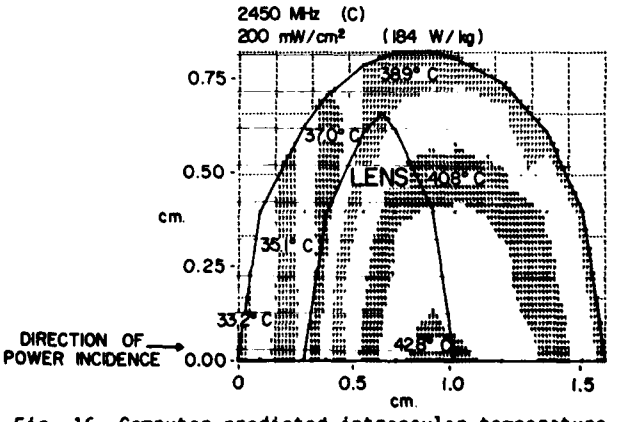


Fig. 16. Computer-predicted intraocular temperature in the rabbit exposed for 30 min at 200 mW/cm² (26)

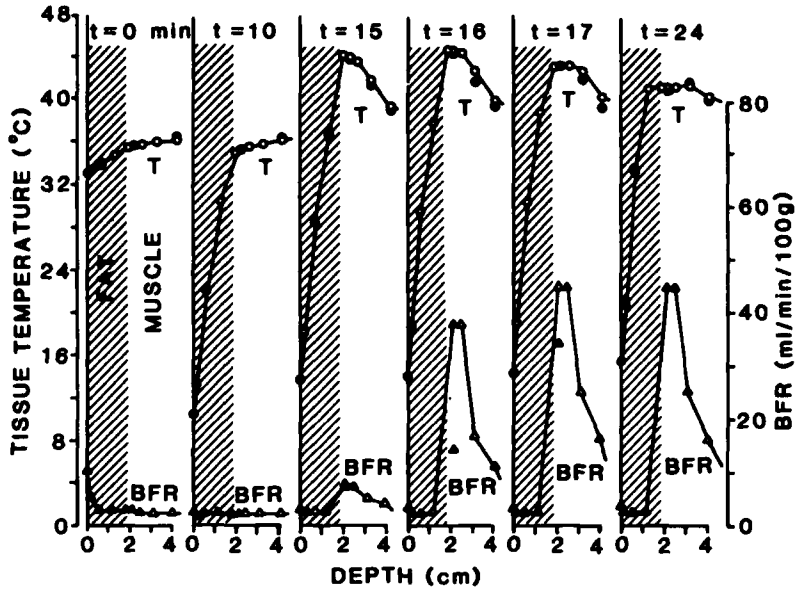


Fig. 7. Time sequence of numerical and experimental blood flow and temperature profiles for exposed thigh. The solid black symbols represent experimental data; open symbols connected by lines represent numerical results. Shading denotes the fat layer (14)

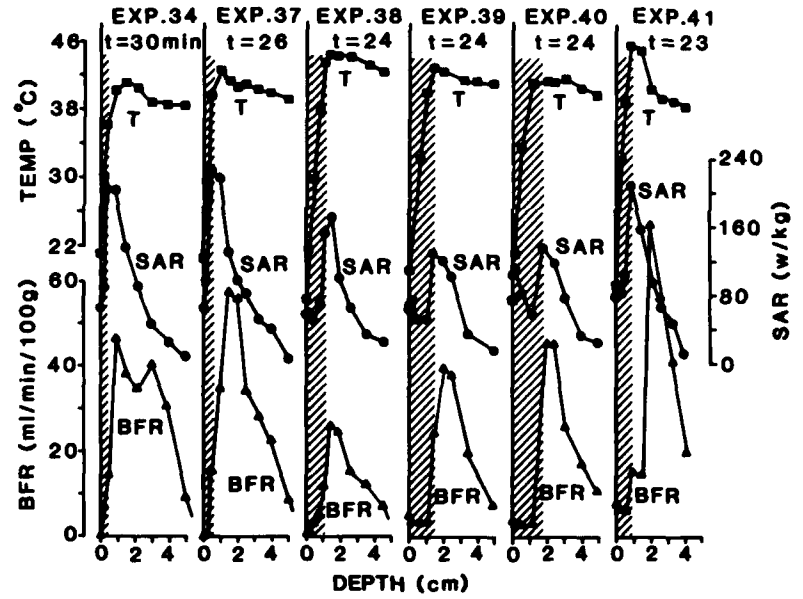


Fig. 8. Numerical values of steady-state temperature, SAR and BFR profiles for various subjects. The shading denotes the fat layer (14)

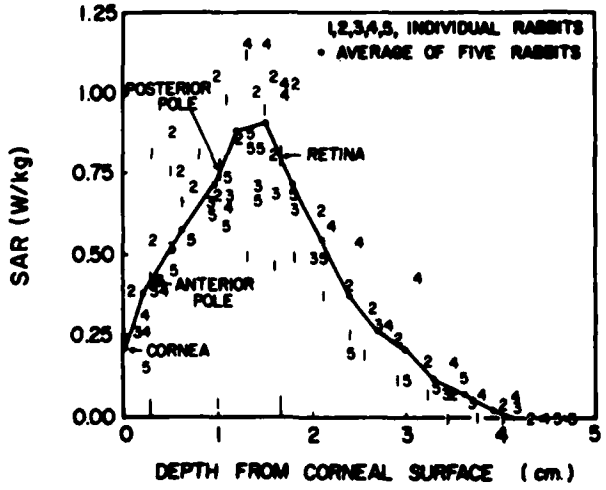


Fig. 9. SAR in rabbit's head and eye exposed to 1 mW/cm^2 microwave radiation (26)

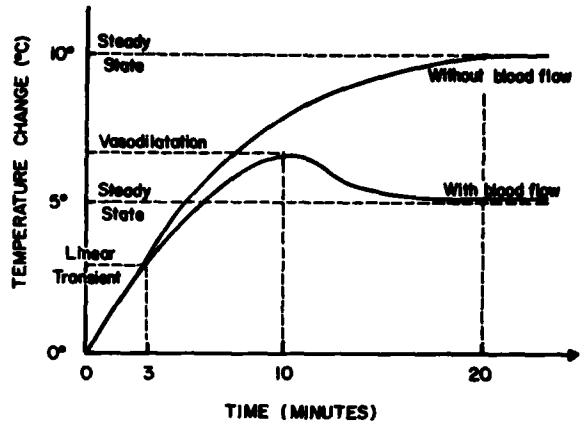


Fig. 1. Transient and steady-state temperature for typical tissue under diathermy exposure (12)

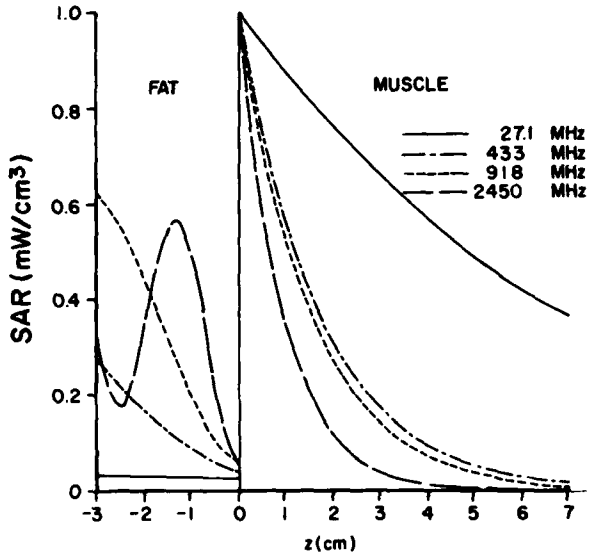


Fig. 2. Relative SAR in planar fat and muscle layers exposed to a wave source (4)

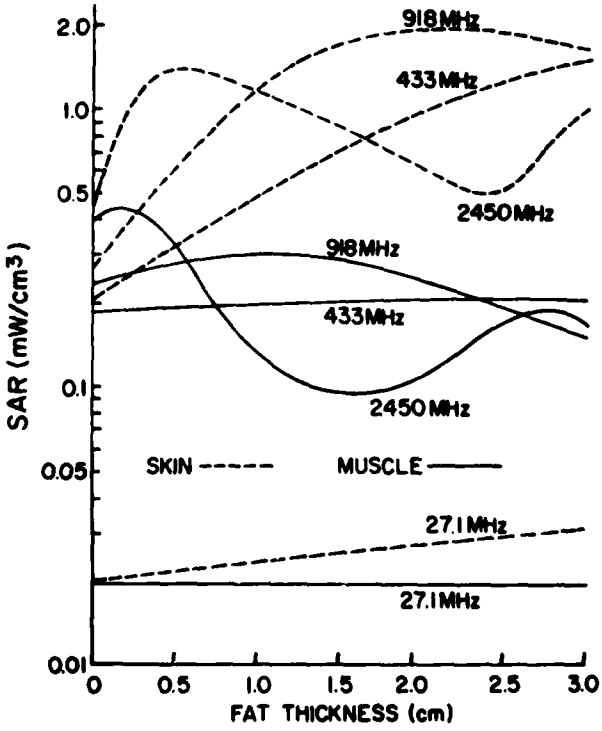


Fig. 3. Peak SAR in planar skin, fat, and muscle layers (4)

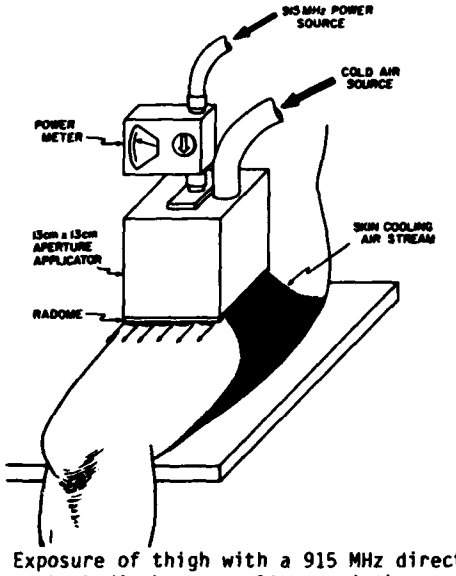


Fig. 4. Exposure of thigh with a 915 MHz direct-contact diathermy applicator (14)

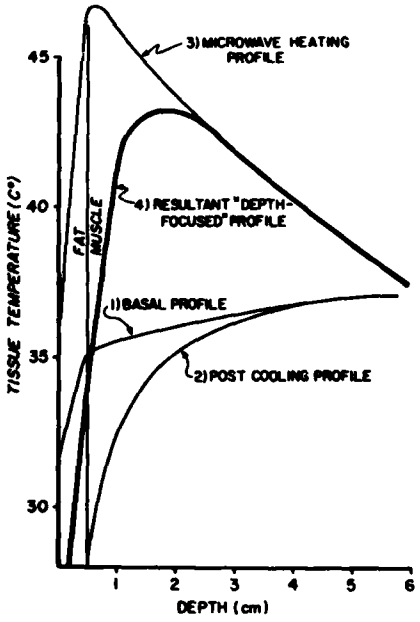


Fig. 5. Temperature associated with diathermy microwave heating and surface cooling (14)

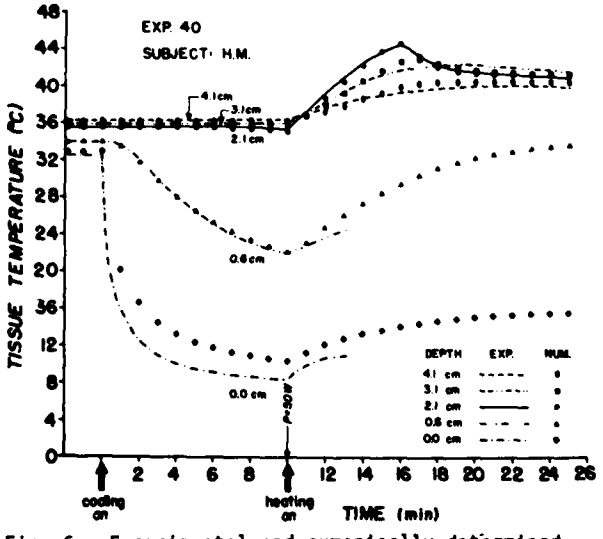


Fig. 6. Experimental and numerically determined transient temperature data for exposed thigh (14)

The Biological Effects of Radiofrequency Radiation

By

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Summary

Exposure of the general public and in particular certain occupational groups to radiofrequency radiation (RFR) is ubiquitous and of growing concern. No clear and widely accepted understanding of the biological effects and health implications of such RFR exposure has emerged. This paper reviews the data available, including reports of RFR effects on single cells or cell components, on genetic composition or development, on developed organs, tissues, or cell systems, and on integrative and regulatory biological systems. Reports of RFR effects on the immunological system, with consideration of the influence of neuroendocrine responses, are critically reviewed in greater detail to illustrate important points regarding data acquisition and assessment, and understanding and application of the RFR bioeffects literature in general. Factors affecting RFR bioeffects research are reviewed, and recommendations for future studies are provided.

Introduction

The general public and especially certain occupational groups are exposed to radiofrequency radiation (RFR) that is ubiquitous and is increasing in regard to sources and potential levels of exposure. Concern regarding such exposures has resulted in numerous studies and anecdotal reports covering many aspects of RFR biological effects. However, no clear and established understanding of RFR bioeffects other than those associated with increased body or tissue temperature has emerged.

Epidemiological studies are discussed in a companion paper. In this paper, we will review the extent of reported experimental RFR bioeffects, using tables to document in brief the vast literature and reported findings. To serve as a basis for generalization, we will critically review, in greater depth, reports of RFR effects on two physiologic regulatory systems which are central to host homeostasis, namely the immunological and neuroendocrine systems. We will discuss the reliability of the reported data and interpretations, with consideration of confounding factors as well as health implications.

General Comments on RFR Bioeffects Studies

Biological systems or structures reported to be affected by exposure to RFR include the neurological, neuroendocrine, hematological, immunological, and cardiovascular, as well as other systems and structure (e.g., ocular lens), cell populations, or subcellular systems or structures. Effects during gestation and early development as well as effects on adults or differentiated cells or tissues have been reported.

There have been so many variations in the biological objects or subjects, as well as methods of investigation, that comparisons between studies, and integration and interpretation of data, are tenuous even if possible. Thus, for example, the literature shows varied targets of exposure (cells, tissues, systems, from different types of animals, etc.), varied exposure conditions (specific absorption rates, ambient conditions, etc., using a wide variety of frequencies and waveforms), and even varied assays for a given bioeffect after a single type of RFR exposure.

There are many identifiable confounding factors, for both animal model studies and experiments with mammalian cells and tissues, which make the relationship of reported RFR bioeffects to the setting of reasonable exposure safety standards at least controversial. Thus, it is necessary to consider pertinent data and concepts relating to (a) animal models versus studies using human cells or tissues, (b) *in vitro* versus *in vivo* studies, (c) environmental conditions and manipulations, (d) single versus repeated exposures, (e) the important roles of heating and stress, and (f) "temperature-independent" versus "non-thermal" bioeffects.

Review of RFR Bioeffects Studies

We have provided an extensive, although still incomplete, list of RFR bioeffect studies in a series of tables, listing the species exposed (or the species from which exposed cells or tissues were derived), the experimental system or model or test specimen, the exposure conditions, the changes in temperature upon exposure (if provided), and the reported effects of exposure. The tables have been adapted (with only minor revisions) from the final report of the US Environmental Protection Agency regarding biological effects of RFR (1).

The tables have been organized to facilitate review of several different overlapping types of potential RFR bioeffects, as follows:

(a) Effects on single cells or cell components
  - Effects on molecular systems (Table 1)
  - Effects on subcellular systems (Table 2)
  - Effects on single cells (Table 3)

(b) Effects on genetic composition or development
  - Genetic and mutagenic effects (Table 4)
  - Teratologic effects (Table 5)
  - Effects on growth and development (Table 6)

(c) Effects on developed organs, tissues, or cell systems
- Effects on testes (Table 7)
- Effects on cardiac function (Table 8)
- Effects on the nervous system (Table 9) and behavioral reactions (Table 10)
- Hematological effects (Table 11)
- Immunological effects (Tables 12 and 13)

(d) Effects on metabolism and on other integrative or regulatory systems
- Effects on clinical chemistry and metabolism (Table 14)
- Neuroendocrine responses (Table 15)

It is impossible within the limits of this paper to fully and critically review such a vast literature regarding RFR bioeffects. General comments regarding such investigations are appropriate at this point and, in the following section, representative reports of immunological and neuroendocrine effects are analyzed in greater detail as examples.

Effects on Single Cells or Cell Components. There are a large number of studies regarding RFR effects on molecular systems (Table 1) and subcellular systems (Table 2), as well as on single cells (Table 3). Most studies of RFR effects on molecular systems have used quite high specific absorption rates (SARs) (Table 1), which are not clearly related in any meaningful fashion to SARs likely to be encountered by humans in the general or occupational environment. This is also true for many but clearly not all of the studies of subcellular systems or single cells.

Effects on molecular systems, when observed, appeared to reflect those changes to be expected from heating, and not specific to the RFR exposure (Table 1). Most studies of the effects of RFR on subcellular systems have shown no changes due to the RFR, with a representative exception being the study by Ismailov (Table 2). Again, the RFR effects were noted at high SARs. RFR has generally not appeared to affect single cells with exposures at low SARs, with occasional exceptions that were possibly related to heating and were reversible (Table 3).

Effects on Genetic Composition or Development. There have been numerous studies regarding possible genetic and mutagenic effects of RFR exposure (Table 4). RFR exposure has resulted in no detectable damage to DNA, or aberrations in chromosomes, or mutations in exposed bacteria, insects, or rodents in many studies. In contrast, other studies have shown changes, such as induction of a repressed protein (not known to be one of the "heat shock" or stress proteins), chromosome aberrations (narrowly frequency-specific), or increased mutations and lethality (Table 4).

RFR-induced teratogenesis has generally followed exposures at very high SARs with few exceptions (Table 5). Occasionally teratogenesis has resulted from exposures at SARs lower than those associated with no adverse effects in other studies using the same model.

RFR exposure has generally not affected growth and development of rodents and primates, although Albert and colleagues have reported RFR exposure-related decreases in rat Purkinje cells (Table 6). Such changes were not noted in those investigators' experiments using monkeys exposed at a slightly higher SAR, although for a slightly varied duration. It thus appears that RFR at low SARs is unlikely to be associated with significant adverse effects on growth and development in most circumstances.

Effects on Developed Organs, Tissues, or Cell Systems. Many studies have been performed to examine potential effects of RFR exposure on developed organs, tissues or cell systems. The cardiovascular and nervous systems, as well as the hematological and immunological systems, have specifically been examined in detail. As with virtually all aspects of RFR bioeffects investigation, the exposure conditions and parameters and methods of measurement have been greatly varied.

Possibly related to the observations noted in the above section are studies regarding RFR exposure of a developed reproductive organ, the testis (Table 7). The only apparent conclusion to be reached from such studies is that exposure to RFR at high SARs can damage testicular tissue, a finding not unexpected in view of the known effects of heating on that organ.

In general, RFR exposure (of the body or just the head) that would result in heating causes increased respiration and heart rate, which might be considered normal physiological responses (Effects on cardiac function, Table 8). While one group reported that synchronized (to the electrocardiogram) RFR pulses produced arrhythmias in frogs, another group reported no effect after attempting to replicate the observation. It has not been established whether the cardiovascular system is affected by exposure to RFR in a manner qualitatively or quantitatively different from other factors or exposures which result in heating.

A major area of interest for investigators of potential RFR bioeffects has been interaction of the RFR with the nervous system (Table 9). Effects reported include alterations of the electroencephalogram, alterations in levels of neuroactive proteins, altered blood-brain barrier permeability, alteration of responses to pharmacologic agents, and morphological damage to neural tissues. Again, exposure conditions have varied widely, but some of the described effects were noted after exposures at relatively low SARs, and occasionally after chronic but not after acute exposures.

Behavioral reactions to RFR have been studied extensively (Table 10), and are often treated as indications of specific neurological effects. While this could be the case in certain instances, in most situations the behavioral response is a normal component of the host's physiological adjustments to maintain thermal (or other) homeostasis. Behavioral reactions have been described after exposures of animals to RFR at relatively low SARs but, as might be expected, reactions were accentuated by exposure at increasing intensities. The environmental conditions, in particular the ambient temperature and humidity, can greatly influence the response to RFR exposure. Even responses to exposure at low SARs, with thermal burdens usually within the range of host physiological adjustment, could be affected significantly by ambient conditions.

Studies regarding effects of exposure to RFR on the hematolical system have measured mainly colony formation by different types of cells, or alterations in circulating (that is, peripheral blood) cell numbers (Table 11). While some studies have shown no effects (commonly with exposures at lower SARs), many have shown alterations in such hematological parameters after exposing several types of animals at moderate to high SARs.

One of the areas of greatest interest in regard to potential RFR bioeffects has been the alleged particular susceptibility of the immunological system to RFR. Many studies have examined immunocompetent cells, or immunological responses after exposure to RFR in vivo (Table 12) or in vitro (Table 13). Lymphocytes, including human lymphocytes exposed in vitro, have been said to be susceptible to RFR, showing enhanced spontaneous proliferation (lymphoblastic transformation) or altered (usually depressed) mitogen- or antigen-stimulated proliferation (a commonly used measurement of immune responsiveness), altered circulation within the body, and other effects of RFR. Many of the early studies reporting such effects on leukocytes were poorly controlled or analyzed, and could not be replicated. Representative studies of RFR effects on the immunological system are discussed in more detail in a subsequent section of this paper, in order to illustrate the difficulties inherent in studying RFR biological effects, to illustrate the errors introduced by improper controls or inadequate analysis of experiments, and to illustrate the potential roles of important confounding factors.

Effects on Metabolism and on Other Integrative and Regulatory Systems. Various effects of RFR on clinical chemistry analyses or on other measurements of the metabolism of animals or exposed animal tissues (commonly brain) have been reported (Table 14).

Of particular importance, potentially affecting all of the above reported interactions of RFR with biological tissues or systems in vivo, is the hormonal response of the host to RFR exposure (Table 15). The neuroendocrine system, now also recognized to be well-integrated with the system of immunological cells and mediators, is a major physiologic regulatory system for maintenance of host homeostasis, responding to RFR exposure as well as other physical or biological challenges. Representative studies, demonstrating neuroendocrine responses to RFR and the effects of such responses on other systems, such as the immune system, are discussed in more detail in the following section.

RFR Effects on Immunological and Neuroendocrine Systems

In this section, selected studies reporting RFR effects on the immunological and neuroendocrine systems will be reviewed in greater depth in order to illustrate important points for critical review, understanding, and application of the RFR bioeffects literature in general. The studies provide examples of pertinent data and concepts relating to (a) animal models versus studies using human cells or tissues, (b) in vitro versus in vivo studies, (c) environmental conditions and manipulations, (d) single versus repeated exposures, (e) the important roles of heating and stress, (f) the interactions of different host physiological and homeostatic systems, and (g) "temperature-independent" versus "non-thermal" bioeffects.

The literature pertaining to RFR effects on the immunological system is extensive and often difficult to interpret and relate to potential risks to humans. Many reports have provided incomplete information regarding experimental design and research methodology (2), or have been anecdotal in character. Where design, methods, and results have been adequately described, they have varied so widely that comparison and overall interpretation remain elusive (2). Many species of animals (and human cells in vitro) have been used, including dogs, rabbits, hamsters, guinea pigs, rats, mice, birds and others. Comparison of studies using different species is difficult, not only due to differential absorption of RFR, but also due to species-specific (and even strain-specific) physiological and immunological responses to a challenge (2,3). Exposures to RFR of 27 to 10,000 MHz, 0.4 to 800 mW/sq cm, with different waveforms (e.g., continuous, or pulsed at a large number of different frequencies), for durations from 5 minutes to years, and under markedly variable environmental conditions have been reported (2). In addition, the immunological functions measured have varied (although less so), either in type of function assayed, or in method of assaying a single function.

The complex nature of the existing data and published reports regarding potential RFR bioeffects can be indicated by consideration of several immunological studies using a common experimental animal, the mouse, for in vivo exposures, as well as several studies using in vitro exposure of human leukocytes. The potential impact of neuroendocrine responses to RFR exposure can also be shown by consideration of the demonstrated interactions between it and the immunological system in exposed animals.

Potential RFR Effects on the Mouse Immunological System. Many animal studies of RFR have used mice or other small rodents. Even with a single species such as the mouse, RFR exposure conditions have varied extensively. Thus, exposure intensities from 0.5 to 450 mW/sq cm, for 5 minutes to more than a year, and under widely varying environmental conditions have been reported (2).

Mice have many disadvantages as well as recognized advantages for studies of RFR bioeffects if the studies are eventually to be used for determining potential effects on humans. The mouse, as a small rodent, has a very different resonant frequency (and thus RFR absorption) compared with that of humans. The mouse has a very different mass-to-surface area relationship, is covered with fur, and has a significantly less efficient thermoregulatory system compared to humans (4-6). Mice remain afebrile or become hypothermic in response to infectious or inflammatory challanges that provoke fever in humans (7-9), even though the mouse has a similar system for production of and response to the host mediator inducing fever, namely endogenous pyrogen (interleukin-1) (6).

On the other hand, since the mouse is a small rodent, many animals can be used within most experimental protocols, allowing properly controlled studies with examination of multiple variables. In addition, the immunological system has been most extensively described for the mouse (not the case for other physiological systems), and inbred strains are available and have been characterized in terms of immunological responsiveness. The latter information allows a more careful examination of the potential effects of exposure to RFR on a particular strain of mouse. However, it is also important to examine

whether any observed effects are strain-specific, as has actually been noted in one series of RFR investigations (3,10).

The neuoendocrine responses of animals to RFR have been studied extensively, mainly using small rodents such as rats, but also using other animals including non-human primates. The acute effects of RFR on hypothalmic-hypophyseal function are generally increased adrenocorticotropic hormone secretion, decreased thyrotropic hormone secretion, and decreased growth hormone secretion (11, 12). Such a pattern of hormone changes constitutes a characteristic and non-specific stress reaction of animals (11). Thus, great care is required in performing RFR experiments to ensure that changes in hormone levels do not result from stress caused by handling of the animals or novelty of the experimental situation (12).

The effects of RFR exposure on endocrine function are generally consistent with both immediate and long-term responses to thermal input and to non-specific stress (which can also be due to thermal loading), although not all investigators agree upon the mechanisms (12). Because of the potential effects of thermal stress, the differences in RFR energy absorption noted above (in regard to the mouse as a model for RFR effects on the human immunological system) may substantially modify the effects of RFR exposure which are mediated by alterations in neuroendocrine function. Notably, there are data which indicate that neuroendocrine function in a primate (such as the rhesus monkey) may be more stable than in a rodent (such as the rat) during thermogenic exposure to RFR at a given frequency (11). Because of the substantial impact of stress and neuroendocrine responses on immunological function, questions regarding RFR exposure effects on the latter system would also arise to the extent that neuroendocrine homeostatic responses are generated by exposure of the experimental animal. It is well documented and widely recognized that whole-body or regional temperature elevations (or decreases) can significantly affect both animal and human immunological functions (2, 13).

Several carefully conducted and well-described studies provide examples of such factors (described above), to be considered in evaluating past results as well as designing future animal investigations.

RFR exposure has been reported to affect expression of a surface marker associated with a stage in maturation of cells of the B lymphocyte lineage. Wiktor-Jedrzejczak et al (14) studied effects of RFR on the quantitative relationships between different splenic lymphoid cell populations of inbred (CBA/J) mice. The mice were exposed, once or three times for 30 minutes each, to 2450 MHz RFR in an environmentally controlled waveguide facility, and their splenic lymphoid populations were compared to those of sham-exposed (control) animals. The specific absorption rates for the exposed animals were 12-15 mW/g. A constant and substantial airflow within the exposure chamber maintained an ambient 25° C temperature. The exposed animals had rectal temperatures after exposure that were from 0.1 to 0.5°C lower than before exposure. Exposures to RFR resulted in an increase in the percentage of cells that expressed complement receptors on their surface (CR+), a characteristic of an intermediate stage of maturing B lymphocytes. Since the increase in CR+ cells was not associated with an increase in the total absolute cell number, it was unlikely that cell proliferation (as opposed to cell differentiation) was responsible for the observed alterations. Further studies also suggested that proliferation of the relevant cell populations was not responsible (15). Several potential mechanisms were considered, but could not be established or disproved by the available data. Furthermore, it could not be determined whether direct or indirect mechanisms (via RFR effects on other interacting systems or cells) were responsible. The cited experiments and subsequent studies by the same investigators are reviewed in greater detail elsewhere (2).

Smialowicz et al (16), noting the above results, performed more extensive investigations of the effects of RFR on lymphocytes from exposed mice of another inbred strain (BALB/c). The mice were exposed to 2450 MHz RFR daily (15 or 30 minutes) for up to 22 days, with specific absorption rates ranging from 4 to 25 mW/g, also in exposure chambers at 22° C with substantial air flow. In contrast to the above studies, they found no differences between exposed and control animals in peripheral blood lymphocyte parameters, such as absolute numbers of specific cell populations, or in frequencies of CR+ B lymphocytes or other types of lymphocytes in the spleen. The parameters of exposure had been chosen to approximate those of Wiktor-Jedrzejczak et al (14). The differences in results were initially suspected to be due to differences in exposure systems: a wave guide system in the studies by Wiktor-Jedrzejczak et al and exposure under far field conditions in the studies by Smialowicz et al. However, subsequently Schlegel et al (10) reported that susceptibility to RFR-induced alterations in percent of splenic cells expressing complement receptors was under genetic control, with mice of one haplotype showing the described alterations but mice of several other haplotypes not demonstrating RFR-induced alterations. In addition, the age of the mice appeared to be critical for the increased expression of complement receptors: mice less than 12 weeks of age did not show altered expression.

In view of the observations of Schlegel et al, Smialowicz et al (17) conducted further studies which confirmed some of the findings, but again showed differing results for some protocols and measurements. Smialowicz et al (17) concluded that many factors were important in determining the variation in appearance of complement receptors, including the age and strain of the mouse, the RFR exposure characteristics (waveguide versus far field), and the environmental conditions. Smialowicz et al had remarked in the earlier report (16) on the absence of colonic temperature changes in RFR-exposed mice relative to sham-exposed animals. Mice exposed at a specific absorption rate of approximately 22 mW/g would have accepted RFR energy equivalent to more than three times their basal metabolic rate. Thus, it is likely that the combination of environmental temperature, humidity, and air velocity in the exposure chamber maintained the mice at their normal temperatures. Further evidence of a thermal burden, with colonic temperature rises abbrogated by the applied environment, was provided by results when mice were exposed as noted above, but without any change of air. Such mice had significantly higher colonic temperatures following exposure.

The above series of investigations demonstrate that choice of animal strains, age of exposed animals, and environmental conditions may all affect results of RFR bioeffects investigations. Another series of studies, by Liburdy, illustrates the potential impact of the interactions between physiological and homeostatic systems such as the immunological and neuroendocrine systems. Liburdy investigated the relationship between RFR exposure and steroid release, associated with thermal stress, and further characterized the nature of potential RFR effects on the immunological system (18, 19).

Liburdy exposed and sham-exposed mice of several inbred strains to 26 MHz RFR at a specific absorption rate of 12.9 mW/g for 4 to 7 minutes, or to warm air (79°C) for 8 to 12 minutes, to induce equivalent increases (2 to 4°C) in rectal temperature (20). Peripheral blood total leukocyte counts were unchanged after exposure to RFR, but there were marked shifts in the proportions of different types of leukocytes. Circulating lymphocytes decreased and circulating neutrophils increased, with pre-exposure values re-established gradually over 55-96 hours. Warm air-exposed and sham-exposed mice both showed an overall leukocytosis, with an increase in both lymphocytes and neutrophils. Liburdy noted that induction of a transient lymphopenia and neutrophilia by RFR exposure was similar in both kinetics and magnitude to changes induced by endogenous release or exogenous administration of adrenocorticotropic hormones. Release of such hormones could be triggered by physical, chemical, or physiological stress. Thus, the differences in circulating leukocytes seen in RFR-exposed versus warm air-exposed animals could indicate an element of stress associated with the rapid heating obtained using RFR, compared to the slower warm air (convective transfer) heating.

Liburdy used two strains of mice (producing equivalent results) in subsequent studies regarding the suspected role of stress, of neuroendocrine response, and specifically of steroid release, in RFR-induced alterations of leukocyte circulation (18). Mice were exposed to 26 MHz RFR (300 mW/sq cm, 5.6 mW/g) or to two alternate hot environments, namely a vented dry air oven (63°C) or a heated water bath (41°C). All exposures were matched to induce a net increase in core body temperature of 2-3°C. Such detectably thermogenic RFR exposures resulted in a peripheral blood lymphopenia and neutrophilia that persisted for 12 hours. Lymphopenia was not induced by alternate exposures to RFR which were not detectably (by changes in core body temperatures) thermogenic (26 MHz, 50 mW/sq cm, or 5 MHz, 800 mW/sq cm, each with a reduction in absorbed power to 0.36 mW/g). Lymphopenia was also not induced by exposures to warm air.

Steroid administration to the mice produced lymphopenia and neutrophilia similar to that induced by obviously thermogenic RFR exposure. Furthermore, Liburdy showed that the thermogenic RFR exposure was associated with a stress-related increase in plasma corticoid levels, with measured levels being threefold greater than those found in sham-exposed or warm-air-exposed mice. Studies with repeated exposures to RFR, including analyses of leukocyte counts in various organs, and including mice to whom exogenous steroids were administered, resulted in similar observations. Alterations in splenic lymphocyte proportions associated with the changes in circulating cells were noted; such changes must be recognized as affecting interpretation of any data derived from studies using only splenic cells from RFR-exposed mice.

Liburdy continued to investigate the effects of RFR exposure on circulation of lymphocytes in vivo, including the relationship of changes in circulatory patterns to steroid release associated with thermal stress and the process of thermoregulation (19). Mice were injected prior to exposure with radiolabelled splenic lymphocytes. At intervals after exposure, sham-exposure, alternate thermal exposure (as above), or steroid administration, various organs were removed and assayed for proportions of labelled cells. Altered lymphocyte traffic was noted with thermogenic RFR exposure (25 mW/sq cm, 19 mW/g) or with steroid administration, but not with thermogenic warm air exposure or with non-thermogenic RFR exposure (5 mW/sq cm, 3.8 mW/g). There were associated changes in immunological responses induced in a particular body location (delayed hypersensitivity measured in footpads) with the altered lymphocyte circulation (after thermogenic RFR exposure) (18, 20).

The thermogenic RFR and warm air exposures both resulted in an increase in core temperature of 2°C within 15 minutes, followed by stable hyperthermia due to adjusted thermoregulation. However, it was noted that although the absolute increases in temperature were identical, the rates of increase were significantly different, with a more rapid increase evident in the RFR-exposed mice. Again, the RFR-exposed mice showed a threefold greater steroid output than the warm air-exposed mice, despite identical final core temperatures. Liburdy suggested that such in vivo whole-body RFR exposure represented a heat stress which stimulated the hypothalmic-hypophyseal-adrenal axis to trigger the release of adrenal steroids into the blood (11,12,21).

Differences in protocols and experimental conditions are apparent when Liburdy's observations (18, 19) are compared to those of Wiktor-Jedrzejczak et al (14). Wiktor-Jedrzejczak et al did not measure peripheral blood leukocyte levels or plasma corticoid levels, and measured rectal temperatures before and after but not during exposure to RFR. Temperature changes in the RFR-exposed mice were minimal, and rectal temperature generally decreased with exposure because of the environmental conditions, namely, forced air convection cooling. The decreases in rectal temperatures occurred despite the fact that the mice were exposed to RFR at twice the specific absorption rate that could lead to an increase (2-3°C) in core temperature (2).

Thus, the environment in the studies by Wiktor-Jedrzejczak et al provided additional (counteracting to the RFR) thermoregulation. The thermal impact of RFR exposure was offset by an environmental hypothermic stress, with likely complex effects (or little net effect) on the animal's physiological regulatory systems (2). The magnitude of thermal load in the experiments of Wiktor-Jedrzejczak et al were likely to have been equal or greater than in the experiments of Liburdy (in which corticoid release was demonstrated in response to an RFR-induced thermal stress). However, the animals in the studies of Wiktor-Jedrzejczak et al appeared to undergo significant cold stress, partially counteracted by RFR absorption (that is, heating). The RFR-exposed animals were thus compared to sham-exposed animals whose physiological regulatory systems were responding to a greater burden (cooling, not offset by RFR) in order to maintain normal core temperatures.

The above studies emphasize the importance of RFR-induced thermal loads, particularly the rate of application of thermal loads, on stress-related neuroendocrine responses as well as the consequent effects of such normal responses to stress on the immunological system. The studies also emphasize the degree to which the environmental conditions for the RFR exposures can influence the findings.

Potential RFR Effects on Human Leukocytes. Studies of potential effects of RFR on human immunocompetent cells have been limited in number and often inadequately described, excessively interpreted, and uncritically cited. Studies by some investigators raised the possibility that human leukocytes are particularly susceptible to RFR (22-24).

An initial (22) and subsequent (23) report of exposure of human mononuclear leukocytes to RFR (2950 MHz) in a 37°C chamber reported both the absence and the presence of a rise in culture temperature using the same protocols. In addition, only limited data were presented resulting from approximately half of the study protocols listed in the methods, which were incompletely described. The mitotic index and percent lymphoblastoid forms in the RFR-exposed leukocyte cultures were said to be different compared with controls, but values from control cultures were not reported, nor were the statistical methods or number of observations.

Another investigator exposed human leukocytes to RFR (2950 MHz) for "various periods, at several power densities" (24). Although that investigator noted that results were poorly reproducible, non-quantitative and only preliminary, RFR-induced lymphoblastic transformation was said to occur. Such results are frequently cited without comment on the inadequate scientific basis for the conclusion (2). In other studies, the investigator also exposed human mononuclear leukocytes to RFR (10,000 MHz) at varying power densities for varying durations(4). Prolonged exposure to RFR, even at low levels ($\leq$ 3 mW/sq cm), caused heating and death of the cells, since culture vessels were not concomitantly cooled. No lymphoblastoid changes were induced by such low-level exposures prior to cell death. In contrast, exposure at power densities from 5 to 15 mW/sq cm, terminated when culture medium temperatures reached 38°C, were reported to induce lymphoblastoid transformation. The initial culture temperatures, the durations of RFR exposure, the rates of temperature increase, and the degrees of change in lymphocyte morphology were not reported.

In view of the above reports, Roberts et al (26) examined the effects of exposure to RFR on human mononuclear leukocytes, measuring viability, DNA, RNA, and total protein synthesis after exposure to RFR (continuous waves) at SARs up to 4 mW/g. They also measured effects on synthesis of specific host defense proteins, namely interferons, and searched for morphological lymphoblastoid transformation as well as changes indicated by incorporation of radiolabelled precursors. Leukocytes were exposed in 37°C chambers without attempts to counteract RFR-induced heating, and final culture temperatures were up to approximately 0.9°C higher than those of sham-exposed cultures, similar to changes induced by exposures in the above-cited studies. Such exposures clearly and reproducibly resulted in no detectable effects on viability or on unstimulated or mitogen-stimulated DNA, RNA, total protein or interferon synthesis by the human mononuclear leukocytes (26).

Since other investigators reported that exposure to pulse-modulated RFR, but not to the unmodulated carrier wave at an equal power density, altered the function of a murine cytotoxic leukocyte line (27), Roberts et al proceeded to examine the effects of pulse-modulated RFR exposure on human mononuclear leukocytes (28). The modulation frequencies implicated in the mouse studies (27), the reported data of which could be criticized on several scientific points (28), included 16 Hz and especially 60 Hz. In addition, some of the earlier studies regarding effects of RFR on human leukocytes used pulse-modulated RFR (23,24). In the studies by Roberts et al (28), exposure of human mononuclear leukocytes to RFR (2450 MHz) pulse-modulated at 16 or 60 Hz, at SARs up to 4 mW/ml, produced no detectable effects on viability or on unstimulated or stimulated DNA synthesis or total protein synthesis. The data provided no evidence that exposure to pulse-modulated RFR is more likely to alter human leukocyte function than is exposure to continuous waves at equivalent energy levels. Such studies, however, do not exclude potential RFR-induced effects on human leukocyte function resulting from exposure at similar SARs, but applied by almost innumerable different possible wave forms (frequencies, modulations, etc.) (26, 28).

Other investigators exposed human mononuclear leukocytes to RFR (27 MHz) for 1-84 hours in a 37°C chamber, in the presence of a mitogenic stimulus, noting a 1°C rise in culture temperature with RFR exposure (25). They noted no differences, compared to controls, in DNA synthesis, cell growth, or mitotic index, but did report an increase in chromosome breaks in RFR-exposed cells after 72 hours of continuous exposure.

The above studies illustrate the importance of adequate description of exposure protocols in research reports and proper statistical analysis of generated data. They also illustrate the controversies that can arise due to the availability and application of innumerable forms of RFR. A continuing controversy about effects of RFR on human (and other) cells and tissues exists in regard to reports of power and frequency "windows", those specific parameters for exposure on either side of which the reported effects are not observed, or are significantly decreased. Frequency "windows", currently emphasized as modulation-frequency-dependent rather than carrier-frequency-dependent, are controversial (28,29), as are power "windows". However, variation in the leukocyte functions which are assayed prevents full assessment of the apparently contrasting observations.

An example of such frequency-specific effects is the reported alteration in protein kinase activity following exposure of cultured human lymphocytes to modulated RFR (29). Byus et al in those studies exposed human tonsil lymphocytes to RFR (450 MHz) amplitude-modulated at 3 to 100 Hz for up to 60 minutes. Since human tonsils are rarely available except when surgically removed from individuals with recurrent or persistent tonsilar inflammation or infection, such results may not reflect upon normal cells, or cells from a site of inflammation under in vivo immunoregulation. (The characteristics of the tonsil donors or of the donated tonsils were not reported).

The modulated RFR fields were applied under conditions in which culture temperature elevations could be expected to be less than 0.1°C. RFR-exposed cells, exposed within a "Crawford chamber", were compared to cells cultured within the same 35°C incubator but external to such a chamber. No data were presented regarding protein kinase activity of human lymphocytes cultured in a Crawford chamber without exposure to RFR (that is, sham-exposed cultures). The kinase activity was reported to remain unchanged in the control cultures, but to be altered by exposure to RFR with certain characteristics, specifically RFR that was modulated at 16, 40, or 60 Hz but not at 3, 6, 80 or 100 Hz and not with unmodulated RFR exposure. The protein kinase that was observed to be affected by the RFR exposure was not cAMP-dependent, and remained to be fully characterized. The kinase activity was reduced transiently, being observed after 15 and 30 minute exposures to RFR, but not after 45 and 60 minute exposures. Although statistical analysis of the data was not reported, the number of observations and the standard errors reported suggested that results could be reproducible.

Such frequency-dependent RFR-induced effects remain to be confirmed by other investigators. The reported RFR-induced effects were transient, and are of uncertain significance even if verified. Nonetheless, they may represent measurable RFR bioeffects, the recognition of which can aid in design of further investigations into extent and health implications of RFR effects on human immunocompetent cells.

Concluding Remarks and Recommendations

Perhaps a major problem for those reviewing the available information regarding potential RFR bioeffects is the perpetuation, via uncritical citation by subsequent authors, of unclear observations or excessive interpretations of data. This perpetuation of unscientific examination of RFR bioeffects may be even more harmful than the original invalid or inappropriately or excessively interpreted investigations. An example of this is the frequently quoted observation of Prausnitz and Susskind (30). Upon ciritical analysis, the study does not at all establish the claimed correlation between exposures of mice to RFR and the development of cancer ("leukemia") (31). In fact, the RFR-exposed mice, despite developing the so-called neoplasms of white blood cells, showed greater survival over the period of the study than did control mice. Nonetheless, because of several considerations, the study neither affirms nor denies a correlation between exposure to RFR and development of cancer, and should not be cited as suggestive of such a correlation (31).

An accurate understanding of RFR interactions with biological systems requires both careful evaluation of past studies and well-designed and well-executed future investigations.

Factors Affecting RFR Bioeffects Research. There are numerous factors which should be considered in the design of future RFR bioeffects investigations. Information regarding such factors should be available to the scientific community in published articles or supplementary technical reports to allow full evaluation of the research findings, as well as replication. Many factors affect RFR absorption, including:

A. Physical parameters of the RFR source:
   1. Frequency
   2. Polarization
   3. Modulation (AM, FM, Pulse, CW)
   4. Power density
   5. Field pattern (Near, intermediate, or far field; Uniformity)
   6. Measuring technique
   7. Calibration technique
   8. Chamber material
   9. Chamber dimensions

B. Biological parameters:
   1. Tissue dielectric properties
   2. Size; geometry
   3. Orientation relative to polarizations
   4. Spatial relations of animals

C. Artifacts:
   1. Ground or conductor plate
   2. Metal implants
   3. Metal or non-metallic objects in the field
   4. Shielding materials

In addition, many factors influence biological responses to the same specific absorption rate, including:

A. Subject variables:
   1. Species; sex; age; weight
   2. Sensitivity
   3. Population density
   4. Interventions (anesthetics; drugs; electrodes; lesions)
   5. Animal husbandry

B. Environmental variables:
   1. Temperature; humidity; air flow
   2. Lighting
   3. Time of day of exposure and sampling
   4. Noise
   5. Odor

C. Concomitant variables:
   1. Genetic predisposition
   2. Base-line of the response
   3. Functional and metabolic disorders

D. Experimental variables:
   1. Acclimation procedures
   2. Duration of exposure
   3. Number and schedule of exposures
   4. Mode of exposure (partial, or whole body)
   5. Sampling technique
   6. Time between exposure and sampling
   7. Restraint devices
   8. Investigator-animal interaction

Several concepts relating to the above factors must be kept in mind. First, biological effects cannot be evaluated as health hazards in isolation. The above discussion notes many but probably not all of the potential confounding factors for investigations of RFR bioeffects. Second, the frequency as well as the form of the RFR may be very important in determining the presence or extent of biological effects. Maximum energy absorption occurs at the body resonance frequency. Also, certain effects have been reported to be strictly frequency-dependent. Third, the distribution of energy deposition may be extremely important. RFR-induced hot spots in critical locations, such as the brain or a local immunological site, may be present and affect results of an investigation despite the absence of elevation in core body temperature. Fourth, a discernable biological effect may not consititute a health hazard. Such a judgement would depend upon many aspects of the personal characteristics of the exposed subject, and would depend very much upon whether the induced perturbation was within or exceeded the limits of the host's ability to compensate, using one or more of the physiological systems available to maintain homeostasis. In fact, some effects may have beneficial applications under appropriately controlled conditions. Examples of the latter

include surgical diathermy and the use of RFR-induced hyperthermia in the treatment of neoplastic diseases (13).

RFR-induced changes should be understood to an extent that is sufficient for determining their clinical significance and their hazard and/or benefit potential, so that appropriate benefit/risk analyses can be applied. It may be important, in assessing the degree of hazard, to determine whether an observed effect is irreversible or irreparable, or is reversible or transient, disappearing after the exposue to RFR is terminated or after some interval of time. However, even a transient effect is to be avoided if it impairs the functional capabilities of the exposed individual, or prevents performance of required tasks.

Recommendations for Future Studies. The criteria which should be able to be used in assessing the results of research on RFR bioeffects include:

(a) The techniques used should assure that possible effects of intervening factors are avoided (e.g., changes in ambient temperature, environmental noise, etc.);
(b) The sensitivity of the experiment should be sufficient to ensure a reasonable probability of detecting an effect if it exists;
(c) The experimental protocols should allow objective collection of data or recording of observations. Wherever the possibility of observer bias exists, methods such as double-blind techniques, with blind scoring or codes, should be used;
(d) All data analyses should be objective and should be subjected to acceptable analytical methods with no relevant data eliminated from consideration;
(e) A given experiment should be internally consistent with respect to the effect of interest;
(f) If an effect is to be claimed, the results should support such an observation at an acceptable level of statistical significance upon application of appropriate tests; and
(g) The results should be quantifiable and susceptible to confirmation by other investigators.

Published articles describing RFR studies should, insofar as possible, address such considerations, within their own texts or, if not editorially feasible, in combination with associated (and available) technical reports.

It should be readily apparent from the above discussions that interactions of RFR with an exposed subject, or even with any individual biological system, such as the immunological system, are complex and difficult to define. Many but not all conflicting observations and interpretations can be ascribed to species specificity and/or varied exposure factors or conditions. Extrapolation from species to species and eventually to humans, in order to formulate safety guidelines for exposure to RFR, requires recognition and definition of multiple confounding factors, as well as a comparative approach with appropriate scaling, or use of proportionality or conversion factors.

Future studies must include a thorough description of methodology as well as results and must include adequate, appropriate controls. Exposure parameters must be reported, not only RFR frequency, waveform, intensity and length of exposure, total dose absorbed and dose distribution, but also environmental conditions.

Continued studies, using both in vivo and in vitro RFR exposure protocols, are needed. It is easier to determine and reproduce the amount of RFR energy absorbed in in vitro experimental protocols. A particular tissue or type of cell or cell system, such as the immunological system, can be examined free of the impact of other potentially exposed cells or systems, or free of diverse concomitant factors or challenges existing in the intact donor. Thus, in vitro studies can often be more tightly controlled.

The exposure of a single biological system independent of other host systems often allows dissection of mechanisms for described effects, unless effects of RFR on the tested system are mediated by another (concomitantly exposed) system. Thus, other physiological and homeostatic systems and interactions will continue to merit consideration. Such "interactive" effects are best recognized using in vivo exposure protocols. Such investigations should include measurements of those biological parameters which allow detection of physiological functions, should identify specific versus nonspecific responses, and should differentiate adaptional or compensatory changes from pathological manifestations.

In vivo investigations may better reflect conditions relevant to determinations of appropriate exposure safety standards insofar as the exposed animal model is representative of human systems. Because of the latter consideration (animal model relevance), and studies showing that animal models are often not directly representative of human systems such as the immunological system (2, 13), one of the most important advantages of in vitro RFR investigation is that studies may be performed using human cells and systems.

Results of exposures of common laboratory animals cannot readily be extrapolated to humans unless a comparative biology approach and some form of "scaling", among animal models and from the animals to humans are used to examine the data generated. Body size of the experimental animal must be taken into account in assessing RFR bioeffects. Body absorption cross sections and internal heating patterns can vary widely. A low-level or suspected "non-thermal" effect may appear to be observed in one animal because the incident power is low, but the animal may actually be exposed to as much absorbed power in a specific (and critical) region of the body as is another larger animal with much higher incident powers. Thus, there is a continuing need, in RFR research as well as in other biomedical investigations, for assessment of the predictive value of animal testing for human responses.

It is important to assess, realistically and without bias, the biomedical effects of exposures to RFR, so that the occupationally exposed worker and the general public will not be unduly exposed to hazards, nor will research, development and beneficial application of forms of RFR be denied or unnecessarily or inappropriately restricted.

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Acknowledgements

The authors' work is supported by contracts or grants from the US Air Force School of Aerospace Medicine (F33615-84-C-0608), the US Department of Energy (DE-AC02-76EV03490), the National Institute of Allergy and Infectious Diseases (AI 15547) and the National Institute of Environmental Health Sciences (ES 03239).

TABLE 1. EFFECTS OF RFR ON MOLECULAR SYSTEMS*

Effects	Experimental System or Model	Exposure Conditions				References
		Frequency (GHz)	Duration (min)	SAR (W/kg)	Modality	
No change in UV difference spectra measured over pH range 2.5-5.5	Bovine serum albumin	1.70 (CW) 2.45 (CW)	30	30-100	Waveguide	32
UV spectra and binding constants for mononucleotides showed no difference from controls	Ribonuclease	1.70 (CW) 2.45 (CW)	30	39	Waveguide	33
No change in enzyme activity	Glucose-6-phosphate dehydrogenase; adenylate kinase; NADPH cytochrome C reductase	2.45 (CW)	5	42	Waveguide	34
No difference in melting curves	DNA	2.45 (CW)	60, 960	67, 160	Far field	35
Inactivation of enzyme; probably temperature inhomogeneity effect at very high doses	Horseradish peroxidase	2.45 (CW)	5, 10, 20, 30, 40	62,500-375,000	Waveguide	36
Heat inactivation of enzymes found at highest SAR (T = 50°C) corresponded closely to heat-treated controls	Glucose-6-phosphate dehydrogenase; lactate dehydrogenase; acid phosphatase; alkaline phosphatase	2.8 (PW)	4.5, 18.5	~200-500	Waveguide	37,38
Heat inactivation of enzyme found at SARs > 165 W/kg	Lactate dehydrogenase	3.0 (CW)	20	33-960	Waveguide	39

*Adapted from EPA (1). CW = continuous wave; PW = pulsed wave.

TABLE 2. EFFECTS OF RFR ON SUBCELLULAR SYSTEMS*

Effects	Experimental System or Model	Exposure Conditions				References
		Frequency (GHz)	Duration (min)	SAR (W/kg)	Modality	
Increase in exchange of strongly bound amide hydrogens in membrane protein measured by infrared spectra for SAR >10 W/kg; no change in α-helix or β sheet content of proteins	Red blood cell membrane	1.0 (CW)	30	5-45	Stripline	40
No change in activity of membrane-bound enzymes measured spectrophotometrically	Red blood cell membrane; mitochondrial inner membrane	2.45 (SW)	10	26	Waveguide	41
No change in activity of membrane-bound enzymes measured spectrophotometrically	Endoplasmic reticulum	2.45 (CW)	5	42	Waveguide	34
No difference in respiratory activity	Mitochondria	2.45 (CW)	30 to 120	17.5, 87.5	Anechoic chamber Far field	42
No difference in respiratory activity	Mitochondria	2-4 (Swept) 3.4 (CW)	10	1.6-2.3 41	Coaxial airline	43
No change in formation of microtubules	Tubulin (Rabbit brain)	3.1 (PW)	15	112-430	Far field	44
No change in migration of proteins within axonal membrane	Vagus nerve cell	3.1 (PW)	24 h	~10-100	Far field	44
No changes in infrared spectra of proteins and nucleic acids in *E. coli* exposed before drying	Dried film of *E. coli* cells	3.2 (CW)	8, 10, 11 h	20	Waveguide	45

*Adapted from EPA (1). SW = sine-wave modulated; CW = continuous wave; PW = pulsed wave.

TABLE 3. EFFECTS OF RFR ON SINGLE CELLS*

End Point Measured or Effects	Experiment System	Exposure Conditions				References
		Frequency (GHz)	Exposure facility	SAR (W/kg)	Duration (min)	
Increase in red blood cell electrophoretic mobility 30 min post-exposure (SAR $\geq$ 10 W/kg)	Red blood cell	1.0 (CW)	Stripline	5-45	4, 8, 15, 30	46
Increase in K^+ efflux and Na^+ influx	Red blood cell	1.0 (CW)	Stripline	45	30	47
K^+ transport no different from heat-treated controls; no change in osmotic fragility	Red blood cell	2.45 (CW)	Monopole far field	3-57	60, 120, 180, 240	48
K^+ transport not different from controls at corresponding temperatures; no difference in hemoglobin release	Red blood cell	2.45 (CW)	Anechoic chamber far field	200	45	49
Passive transport of Na^+ and Rb^+ increased at transition temperature	Red blood cell	2.45 (CW)	Waveguide	100, 190, 390	60	50
No significant changes in K^+ efflux, hemoglobin release, or osmotic fragility	Red blood cell	2.45 (CW) 3.00 (CW) 3.95 (CW)	Waveguide	22-200	20, 180	51
Rapid response in change of firing rate of pacemaker neurons which does not correlate with temperature changes in minority of trials	Isolated neuron from *Aplysia*	1.5, 2.45 (CW and PW)	Stripline	1-100	3	52,53
No change in growth or CFU of exposed cultures	*E. coli* *Ps. aeruginosa*	2.45 (CW)	Far field	29-320	720	35
No change in growth or CFU of various strains of exposed cultures under several growth conditions	*E. coli*	2.45 (CW)	Anechoic chamber - far field	0.0075-75	240	54
No change in survival curves (measuring CFU) of exposed cultures	*E. coli* *B. subtilis* spores	2.45	Microwave oven	~400	1	55
Growth rate slowed; morphological changes found	Chinese hamster lung cells, V79	2.45 (CW)	Waveguide	1059	20	56
No change in light emission of photoactive bacterium	*P. fischeri*	2.6-3.0 (CW)	Waveguide	660-5300	~22	57
No effect on colony-forming ability	*E. coli*	2.6-4.0 (CW)	Waveguide	29	8 h	45
Temporary decrease in virulence (>6 h) of bacteria for its host cells; recovery within 24 h at 37°C	*A. tumefaciens*	10 (CW)	Cavity	~1	30, 60, 230	58

*Adapted from EPA (1). CW = continuous wave; PW = pulsed wave; CFU = Colony forming unit.

TABLE 4. GENETIC AND MUTAGENIC EFFECTS OF RFR EXPOSURE*

Effects	Test Specimen	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/m²)	SAR (W/kg)	Duration (days X min)	
No change in thermal denaturation profile, except at elevated temperature	DNA	2.45 (CW)	1340	67 (est)	1 X 960	35
Change in thermal denaturation profile and hyperchronicity of DNA extracted from testes following exposure	Mouse	1.7 (CW) 0.985 (CW)	≤500 100	≤2.4 (est) ≤0.26 (est-testes)	1 X 80	59,60
No chromosome aberrations in white blood cells	Chinese hamster	2.45 (CW)	50-450	21	5 X 15	61
No sister chromatid exchange in bone marrow cells	Mouse	2.45 (CW)	200	21	28-480	62
No chromosome aberrations in CHO-K1 cell line if temperature maintained	Chinese hamster	2.45 (CW)	≤2000	≤360 (est)	1 X 30	63
No chromosome aberrations or change in mitotic activity in regenerating liver cells in rat	Rat	0.013 (CW) 0.013 (PW)	(4.45 $kV_{p-p/m}$) (44.1 $kV_{p-p/m}$)	1.3 (est)	1 X 1,680-2,640	64
No mutation induction	*E. coli* *E. coli*	2.45 (CW) 1.7 (CW)	100 or 500 (250 $V_{p-p/m}$)	15 or 70 3	1 X 180-240 1 X 120	65
No mutation induction observed in Ames tester strains	*S. typhimurium*	2.45 (CW) 8.6-9.6 (PW)	200 100, 450	40 18, 80 (est)	1 X 90 1 X 90	66
Reduction in survival concomitant with rise in sample temperature	*E coli* *S. typhimurium* *S cerevisiae*	8.6-9.0 (PW) 8.6-9.0 (PW) 8.6-9.0 (PW)	10-200 ≤450 ≤450	≥50 ≥80 ≥80	1 X 90 1 X 90 1 X 120	67
No reduction in survival or mutational events	*S. cerevisiae*	70.5 (CW) 73 (CW)	≤600	≤17 (est)	1 X 180	68
No reduction in survival or mutational events	*S. cerevisiae*	9.4 (CW) 17 (CW)		≤28 (est)	1 X 300 1 X 1440	69
No detectable lethal events due to no change in colony-forming units	*E. coli*	2.6-4.0 (CW)		19	1 X 480	45
No observable change in molecular structures because no change in infrared spectrum	*E. coli*	3.2 (CW)		21 or 16	1 X 660-720	45
No repairable DNA damage	*E. coli*	8.6 (PW)		12	1 X 60-420	70
No change in growth pattern; enhanced *colony-forming* activity	*E. coli*	2.45 (CW)	.05-500	0.008-75	1 X 240	54
No change in mutation frequencies at either of two loci controlling requirements for adenine and tryptophan	*S. cerevisiae*	2.45 (CW) 8.5-9.6 (PW)	200 10-450	40 ≤80 (est)	1 X 120 1 X 120	66
No mutagenic effects in exposed embryos	*D. melanogaster*	2.45 (CW)		100	1 X 360	71
No changes in generation time, sex ratio, nor sex-linked lethal mutations in offspring	*D. melanogaster*	2.45 (CW)	46,000-65,000	150-210 (est)	1 X 45	72
No mutations in adult males as evidenced by chromosome loss; nondisjunction; nor sex-linked recessive lethals	*D. melanogaster*	0.029 (CW) 0.146 (CW)	(600 $V_{rms/m}$) (62.5 $V_{rms/m}$)	0.024 (est) 0.015 (est)	1 X 720 1 X 720	73
No mutagenic changes (recessive lethals) in adult females	*D. melanogaster*	0.098 (CW)	(0.3 $V_{rms/m}$, (FM) at audio)	0.0004 (est)	224 X 1140	74
No significant germ-cell mutagenesis in weekly breedings	Rat	2.45 (CW)	50	4.7-0.9	106 X 240	75
No significant germ-cell mutagenesis in weekly breedings	Rat	2.45 (CW)	100	2	5 X 300	75
Same, except decrease in pregnancies, indicating temporary sterility caused by elevated testicular temperatures	Rat	2.45 (CW)	280	5.6	20 X 240	75
Induction of a repressed protein, collicin, indicating a change in the genetic processes	*E. coli*	37 (CW)	0.01-10		1 X 30-120	76
Change in growth rate that was very frequency specific, indicating an alteration in the processes of the cell	*S cerevisiae* *S.cerevisiae*	41-42 (CW) 41.650-41.825 (CW)	10-30 ≤100	4-11 (est. average)	1 X 660 1 X 180	77
Chromosome aberrations in lung cells in vitro at two frequencies but not at two closely related frequencies, 0.015 or 0.025 GHz	Chinese hamster	0.019 (PW) 0.021 (PW)	(up to 300 $kV_{p-p/m}$)		1 X 30	79,80
Increase in chromosome translocations in sperm cells	Mouse	9.4 (PW)	1-100	0.05-5 (est)	10 X 60	81
Increased mutations and lethality	*S. typhimurium*	2.45 (CW)	51,000		1 X 0.03-0.48	82
No change in growth when compared to temperature controls	*E. coli*	8.8 (PW)		12	1 X 900	83
No change in survival or mutation induction	*E. coli*	9.4 (CW) 17 (CW) 70-75 (CW)		23 28 9	1 X 30 1 X 30 1 X 30	84
	S. cerevisiae	9.4 (CW) 17 (CW) 70-75 (CW)		23 28 28	1 X 30-2,880 1 X 30-2,880 1 X 30	
No change in dominant lethality	Mouse	2.45 (CW)		43	1 X 30	85
No change in mutation induction	*E. coli*	0.027 (CW) 2.45 (CW) 3.07 (PW)		≤4 35-100 35-100	1 X 60-400 1 X 60-400 1 X 60-400	86
	S. Typhimurium	0.027 (CW) 2.45 (CW) 3.07 (PW)		≤4 35-100 35-100	1 X 60-400 1 X 60-400 1 X 60-400	
Higher mutagenicity index perhaps due to heating and RFR	Mouse	2.45 (CW) 2.45 (CW) 1.7 (CW)	1000 500 100	11.4 (est-testes) 5.7 (est-testes) 0.5 (est-testes)		59

*Adapted from EPA (1). CW = continuous wave; PW = pulsed wave; est = estimated; est-testes = estimated for testes only.

TABLE 5. TERATOLOGIC EFFECTS OF RFR EXPOSURE*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/kg)	SAR (W/kg)	Duration (days X min)	
30% survival of pupae	*D. melanogaster*	2.45		640	1 X 10	87
Embryonic LD_{50} (exposure of egg)	Chicken	2.45	2000	70	1 X 12	88
			2800	98	1 X 7	
			4000	140	1 X 4	
Decreased postnatal survival	Mouse	2.45		104	1 X 4	89
Teratogenesis	Mouse	2.45		85-112	1 X 2-5	90,91
No change in teratogenesis	Mouse	2.45		38	1 X 10	92
Increased postnatal survival	Mouse	2.45		38	1 X 10	92
Maternal lethality, resorptions decreased fetal weight	Rat	2.45		31	1 X 20	93
Decreased fetal weight	Mouse	2.45	280	22	12 X 100	94
No change in hatchability, post-hatching hemogram, body or organ weights (exposure of egg)	Japanese quail	2.45		14	1 X 1440	95
No change	Rat	2.45		14	1 X 20	96
No change	Rat	2.45	400	10	1 X 120	97
No change	Mouse	2.45	34-140	2-8	12 X 100	94
Teratogenesis (exposure of egg)	Japanese quail	2.45		4	12	98
No change	Rat	2.45	50	0.7-4.7	"Many" X 240	99
No change	Rat	2.45	280	4.2	12 X 100	100
No change	Squirrel monkey	2.45		3.4	"Many" X 180	101
No change	Rat	0.914, 2.45, 6	100-350	1-3.5	"Many"	102,103,104
No change	Rat	0.918	50	2.5	19 X 1200	105
Decreased body and brain weight	Rat	2.45	100	2.2	16 X 300	106

*Adapted from EPA (61). SAR from report or estimated (1).

TABLE 6. EFFECTS OF RFR ON GROWTH AND DEVELOPMENT*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/cm^2)	SAR (W/kg)	Duration (days X min)	
No effect on weight gain	Rat	2.45 (CW)	50	0.7-4.7	55 X 240	99
No effect on growth, neurological or immunological development or mutagenicity	Rat	0.1 (CW)	460	2.8	112 X 240	107
Possible decrease in brain acetycholinesterase activity					37, 55 X 240	
Decrease in Purkinje cells	Rat	0.1 (CW)	460	2.8	112 X 240	108
Decrease in Purkinje cells	Rat	2.45 (CW)	100	2	5 X 1260	
Decrease, then recovery in Purkinje cells	Rat	2.45 (CW)	100	2	5 X 240	
No change in Purkinje cells	Monkey	2.45 (CW)		3.4	285 X 180	109
No change in infant mortality	Monkey	2.45 (CW)		3.4	285 X 180	101
No effect on growth	Mouse	2.45 (CW)	100	6-8 (est)	24 X 48	110
No effect on body weight of infant rats	Rat	2.45 (CW)	400	20-60 (est)	6 X 5	111
No effect on growth	Mouse	0.01, 0.019 0.026 (CW)	89,000	0.9, 1.8, 3.6 (est)	1 X 20	112
	Mouse	0.019	1.7×10^5 11.4×10^5	6.3 (est)	5 X 40	
No effect on weight gain	Mouse	0.148 (CW)	5	0.013	50 X 60	113

*Adapted from EPA (1). CW = continuous wave; est = estimated.

TABLE 7. EFFECTS OF RFR ON TESTES*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration (days X min)	
No change	Mouse	1.7	100	15	1 X <100	114
Abnormal germinal cells, normal interstitial cells			100	15	1 X 100	
All tissue necrotic			500	75	1 X 30-40	
Scrotal skin burns			2000	300	1 X 20	
"Minimal" injury	Mouse	3.0	500	50	1 X 20	
No change in tissue, sperm	Mouse	2.45	<370	<8	"many" X 16 h	115
Abnormal spermatogenic tissue	Rat	2.45	800	16 16	1 X 10-73 5 X 10-73	116
No change	Rat	2.45	50	0.9-4.5	"many" X 240	75
No change			100	2	5 X 360	
Temporary sterility			280	5.6	20 X 240	

*Adapted from EPA (1).

TABLE 8. EFFECTS OF RFR ON CARDIAC FUNCTION*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Integsity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Bradycardia develops after whole-body exposure, along with hyperthermia	Rat	2.45 (PW)	280, 480	6.5, 11.1	1 X 30	117
Exposure to head promotes tachycardia; exposure to back raises respiratory rate but not heart rate	Rabbit	2.4 (CW, PW)	200	3	1 X 60	118
Increased respiration	Rabbit	2.4	400-1000	8-20 (est)	1 X 20	119
Increased heart rate from dorsal exposure of the head	Rabbit	2.4	1000	20 (est)	1 X 20	119
Alterations in electrocardiogram (shortening of QT interval, increased height of T-wave, appearance of U-wave) of 72 h chick heart	Chicken	24(PW)	740		1 X 3	120
No effect on heart rate of embryo that cannot be attributed to microwave heating	Quail	2.45 (CW and PW)		0.3-30	1 X 5-10	121
Pulses synchronized with each R-wave do not affect heart heart rate	Frog	1.42-10 (PW)	0.32		(100-μs pulses)	122
Synchronized pulses with QRS complex causes increase in heart rate with some arrhythmias	Frog	1.425 (PW)	.006		(10-μs pulses)	123
Increased heart rate	Rabbit	2.45 (CW)	800	12	10 X 20	124
No effect on heart rate			50	0.3, 0.093	10 X 20	
Low power levels cause bradycardia in the isolated heart	Turtle	0.960 (CW)		2-10	1 X 60	125
Causes slight decrease in the isolated heart rate	Rat	0.960 (CW)		1.3, 2.1	1 X 5-10	126
Exposures synchronized with electrocardiogram have no effect on heart rate	Frog	1.42-3 (PW)	0.006		(2-, 10-, 150-μs pulses)	127

*Adapted from EPA (1). PW = pulsed wave; CW = continuous wave; est = estimated.

TABLE 9. EFFECTS OF RFR ON THE NERVOUS SYSTEM*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Integsity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Desynchronized EEG	Rabbits	3 (PW)	200 (av)	3.0 (est)	1 X 20	128
Greater effect of CNS-stimulating drugs	Rabbits	3 (PW)	70 (av)	1.0 (est)	24-26 X 180	128
Biphasic effect of latency to a convulsive drug effect	Mice	3 (PW)	50	5	8-36 X Unknown	129
Decreased effect of paralyzing drugs	Rats	3 (PW)	50	1	10-15 X Unknown	129
Changes in EEG patterns of unanesthetized animals	Rabbits	9.3 (CW)	7-28	0.1-0.3 (est)	1 X 5	130
Potentiation of drug response	Rats	2.45 (PW)	10 (av)	0.2 (est)	1 X 30	131
Decreased hypothalamic NE, DA, and hippocampal serotonin in hyperthermic animals	Rats	1.6 (CW)	800	24 (est)	1 X 10	132
Decreased hypothalamic NE, DA	Rats	1.6 (CW)	200, 800	6-24 (est)	1 X 10	133
No effect on neurotransmitter levels	Rats	1.6 (CW)	100	3.0 (est)	1 X 10	133
No effect on GABA content	Rats	2.86 (PW)	800 400 100 100	16.0 (est) 8.0 (est) 2.0 (est)	1 X 5 1 X 20 5 X 480 40 X 240	134
Swollen neurons in hypothalamus and subthalamus	Chinese hamsters	2.45 (CW)	500 250	15 (est) 7.5	1 X 30 22 X 840	135
Swollen neurons in hypothalamus and subthalamus	Chinese hamsters	1.7 (CW)	100	3 (est)	1 X 30-120	136
Myelin figures in dendrites 6 weeks post-exposure of females	Rats	2.45 (CW; multimodal cavity)	100	2.3	110 X 300	137
Increased permeability of BBB to fluorescein	Rats	1.2 (CW) 1.2 (PW)	24 2 (av)	1.0 (est) 0.08 (est)	1 X 30 1 X 30	138
Myelin degeneration and metabolic alterations; glial cell proliferation	Guinea pigs, Rabbits	3 (CW;PW) 3 (CW;PW) 3 (CW;PW)	35 250 50	0.5 (est) 3.5 0.4 (est)	90 X 180 1 X 180 90 X 180	139
Focal areas of increased BBB permeability to peroxidase	Chinese hamsters, Rats	2.8 (CW) 2.8 (CW)	100 100	1.9 0.9	1 X 120	140
Increased peroxidase in brain, absent after recovery period	Chinese hamsters	2.45 (CW)	100	2.5	1 X 120	141
Increased peroxidase in brain	Chinese hamsters	2.45 (CW)	100	2.5	1 X 120	142
Brain temperature elevation (40-45°C); increased permeability of BBB	Rats	2.45 (CW)	(80 W)	-	1 X 10-30	143
Increased permeability of BBB (mannitol and inulin)	Rats	1.3 (CW) 1.3 (CW)	10 3 (av)	0.4 0.1	1 X 20 1 X 20	144
Decreased latency of late components of thalamic somato-sensory evoked potentials	Cat	0.918 (CW)	26	2.5	1 X 15	145
Attenuation of monosynaptic spinal reflex	Cat	2.45 (CW)	37.5	800	1 X 3	146
EEG effects seen after chronic but not acute exposures	Rabbit	0.001-0.01 (AM) 0.001-0.01 (AM)	(60-500 V_{rms}/m) (90-500 V_{rms}/m)	10^{-5}-10^{-4} 10^{-4}-10^{-3}	1 X 120-180 20-30 X 120-180	147
Change of predominant EEG frequencies	Cat	0.147 (AM)	10	0.015 (est)	Varying	148
Reversible neuronal morphology alterations	Rat Rat	3 3	100 100	2 (est) 2 (est)	35 X 30 35 X 30	149 150
Mild pyknosis of hippocampal neurons, increased brain and colonic temperature	Rat	2.45	(60-90 W)	head only	1 X 2.5-7	151
Increased brain serotonin turnover rate	Rat	3	400	8.0 (est)	1 X 60	152
Decreased brain serotonin turnover rate	Rat	3	100	2.0 (est)	7 X 480	152
No decrease in cerebellar purkinje cells in offspring	Squirrel monkey	2.45	100	3.4	368 X 180	108
Decreased cerebellar purkinje cells after perinatal exposure	Rat	2.45 0.100	100 460	2.0 2.7	5 X 1260 110 X 240	109

*Adapted from EPA (1). PW = pulsed wave; CW = continuous wave; av = average; est = estimated; AM = amplitude modulation; NE = norepinephrine; DA = dopamine; BBB = Blood-Brain Barrier; EEG=electroencelphalogram; CNS = central nervous system; GABA = gamma-aminobutyric acid.

TABLE 10. BEHAVIORAL REACTIONS TO RFR*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Decreased exploratory activity and swimming speed, ΔT increase = 2.5°C	Rat	2.45 (PW, multimodal cavity 120 Hz, AM)	?	6.3	1 X 30	153
No effect on spontaneous activity nor forced running	Rat	10.7 (CW) 3 (CW) 3 (PW) 3 (PW)	6-9 5-10 15-20 240-260 (av)	0.2 0.3 0.6 8.3	7.7 x 1440 7.7 x 1440 7.7 X 1440 17 X 1440	154
Increased locomotor activity	Rat	2.45 (CW, multimodal cavity)	100	2.3	110 X 300	155
Decreased spontaneous activity and food intake	Rat	0.918 (CW)	100	3.6	21 X 600	156
No effect on spontaneous activity nor food intake	Rat	0.918 (CW)	25	1.0	91 X 600	157
Decreased activity on stabilimetric platform, no significant increase in wheel running	Rat	2.45 (CW)	50	1.2	80 X 480	158
Increased activity on stabilimetric platform and in wheel running	Rat	0.915 (CW)	50	2.5	80 X 480	159
Decreased time on treadmill and inclined rod, decreased exploratory activity, increased then decreased shock sensitivity. Decreased activity and shock sensitivity persisted 90 days after exposure	Rat	2.375 (CW)	5	0.1	30 X 420	160
Colonic temperature rise = 0.37°C before start of test; ΔT = 1.5°C with microwaves	Rat	2.45 (PW, multimodal cavity, 60 and 12 Hz AM)		420 220	10 X 0.17 10 X 0.5	161
Response decreased during exposure on random interval schedule (lowest intensity for effect, ΔT = 1.8°C)	Rat	0.500 (CW)	250	10	1 X 11	162
Response decreased during exposure (maximum effect) on random interval schedule, ΔT = 1.8°C	Rat	0.600 (CW)	100	7.5	1 X 55	163
Decreased observing responses on vigilance task, ΔT = 2°C	Rhesus monkey	2.45 (120 Hz, AM)	720	5.0	1 X 60	164
No effect on observing responses			160	1.1	1 X 20	164
Decreased observing responses on vigilance task	Squirrel monkey	2.45 (120 Hz, AM)	500	2.8	1 X 30 1 X 60	165
No effect on observing responses				0.6-1.7	1 X 60	165
Decreased observing responses on vigilance task	Rat	1.28 (PW) 5.62 (PW)	100 260	2.5 4.9	1 X 40 1 X 40	166
Response rate decreased on fixed interval schedule in rats with high base-line rates, spending time away from lever	Rat	2.45 (120 Hz, AM)	375	7.5	1 X 60	167
No effect on response rate			88-184	1.8-3.7	1 X 60	167
No effect on visual tracking task	Rhesus monkey	1.2(CW)	200	1.6	1 X 120	168
Response rate decreased on FR and increased on DRL schedules	Rat	2.45 (CW) 2.86 (PW) 9.60 (PW)	50 50 50	1.4 1.4 1.5	1 X 30 1 X 30 1 X 30	169
Decreased length of runs and fewer reinforcers on FCN schedule	Rat	2.45 (CW)	50	?	1 X 30	170
Decreased response rate on FR operant schedule	Rat	2.45 (CW)	100	2.7	. X 900	171
Increased rate of missing observing responses on vigilance task	Rat	2.45 (PW, AM, multimodal cavity)		6.5	1 X 30	153
Decreased rate of responding on repeated acquisition task	Rat	2.80 (PW)	50	0.7	1 X 30	172
Increased response rates in extinction, decreased stimulus control, no effect on Sidman avoidance	Rat	2.45 (CW, multimodal cavity)	100	2.3	110 X 300	155
No effect on flavor aversion test	Rat	0.918 (CW)	100	3.9	21 X 600	156
No effect on flavor aversion test	Rat	0.918 (CW)	25	1.0	91 X 600	157
Microwaves detected as stimulus	Rat	2.45 (PW 120 Hz, AM, multimodal cavity		0.6-2.4	1 X 1	173
Microwaves detected as stimulus	Rat	0.918 (PW)	150	7.5	1 X 0.5	174
Spending more time in shielded vs. unshielded compartment	Rat	1.20 (PW)	2	0.2	4 X 30	138
Spending equal time in shielded vs. unshielded compartment	Rat	1.20 (CW)	24	2.2	4 X 30	138
Spending more time in shielded vs. unshielded compartment (occurred in first of 7 sessions)	Rat	1.20 (PW)	4	0.4	1 X 90	175
Spending more time in unirradiated compartment	Rat	2.80 (PW)	95	2.1	9 X 60	176

Table 10, continued

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Decrease in SAR at 24°C	Mouse	2.45 (CW)		28	1 X 15	177
Decrease in SAR when ambient temperature increased from 20°C to 35°C	Mouse	2.45 (CW)		43.6-0.6	1 X 20	178
No preferential orientation of rats or mice in far field of plane wave	Rat	2.45 (CW)	150	3.3	1 X 60	179
	Mouse	2.45 (CW)	150	6.2-12.3 (depending on orientation)	1 X 60	
Cannot take specific action to reduce intensity of irradiation	Rat	0.918 (PW, 60 Hz, AM, multimodal cavity)		60	5 X 2	180
Augmentation of increased response rate: produced by chlordiazepoxide	Rat	2.45 (PW)	10	0.2	1 X 30	131
Shift to lower doses of dose-response curve for d-amphetamine in DRL schedule	Rat	2.45 (PW)	10 10	0.2 0.2	1 X 30 4 X 30	181
No effect on dose response curve for chlorpromazine or diazepam	Rat	3.8 (PW)	10	0.2	1 X 30	182
Chlordiazepoxide reduced responses, decreased avoidance responses, and increased escape responses to RFR	Mouse	2.45 (CW)		46	1 X 30	183
Response rate decreases were augmented after exposures at higher ambient temperatures	Rat	2.45 (CW)	100	2.0	1 X 930	184
Reduced responding for heat lamp in a cold room	Rat	2.45 (CW)	50	1.0	1 X 15	185
Selection of a lower ambient air temperature	Squirrel monkey	2.45 (CW)	60	1.0	1 X 10	186

*Adapted from EPA (1). If measured SAR was not reported, SAR was estimated when possible. PW = pulsed wave; CW = continuous wave; AM = amplitude modulated; av = average.

TABLE 11. EFFECTS OF RFR ON THE HEMATOLOGICAL SYSTEM*

Effects	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Increased: WBC, Ly, PMN, RBC, Hct, and Hgb	Rat	24 (PW)	100 200	3.0 (est)	1 X 180 1 X 420	187
No change	Dog	24 (PW)	240	1 (est)	400 X 400-900	188
No change Decreased: RBC, WBC and Ly Increased: PMN	Rat	3 (PW)	100 400 1000	2 (est) 8 (est) 20 (est)	216 X 60 20 X 15 6 X 5	189
Decreased: Ly and Eos Decreased: WBC, PMN, and Eos Decreased: WBC, Ly and Eos Increased: PMN Decreased: Ly Increased: PMN	Dog	2.8 (PW) 2.8 (PW) 1.28 (PW) 0.200 (CW)	1000 1650 1000 1650	4 (est) 6 (est) 4.5 (est) 25 (est)	1 X 360 1 X 120 1 X 360 1 X 360	190
No change	Mouse	0.800	430	12.9 (est)	175 X 120	191
Increased: Ly and mitotic index of lymphoid cells	Guinea pig	3 (CW or PW)	35	0.5 (est)	120 X 180	192, 193
Increased: RBC, Hct, and Hgb	Rat	2.4 (CW)	100	2 (est)	30 X 120	194
No change	Rat	2.4 (CW)	50	1 (est)	90 X 60	195
Increased: Eos	Rabbit	2.45 (CW)	100	1.5	180 X 1380	196
Increased: WBC, CFU Decreased: ^{59}Fe uptake	Mouse	2.45 (CW)	1000	70 (est)	1 X 5	197
Accelerated recovery following X-irradiation; increased erythropoiesis and myelopoiesis	Mouse	2.45 (CW)	1000	70 (est)	1 X 5	198
Accelerated recovery from X-irradiation	Dog	2.8 (CW)	1000	4 (est)	1 X 3600	199
Increased: PMN and RBC Decreased: Ly Accelerated recovery from X-irradiation	Chinese hamster	2.45 (CW)	600	28 (est)	1 X 30	200
Increased: Ly Decreased: PMN (Not reproduced consistently; perinatal exposure)	Rat	0.425 (CW)	100	3-7	47 X 240	201
No change (perinatal exposure)	Rat	2.45 (CW)	50	1-5	57 X 240	99
No change (perinatal exposure)	Rat	0.1 (CW)	460	2-3	57 X 240	107
No change (exposure of egg)	Quail	2.45 (CW)	300	14	1 X 1440	95
Decreased: Hct, WBC, and Ly	Rat	2.736 (PW)	244	5-25 (est)	35 X 240	202
No change	Mouse	2.45 (CW)	300	22	22 X 30	13
Decreased: Ly Increased: PMN	Mouse	0.026 (CW)	86,100	13 (est)		20
Decrease in CFU for erythroid and granulocyte-macrophage series	Mouse	2.45 (CW)	150	10	9 X 30	203
Reduction in CFU granulocyte-macrophage precursors exposed *in vitro*	Mouse	2.45 (CW)	600-10,000	120-2000	1 X 15	204

*Adapted from EPA (1). WBC = white blood cell; Ly = lymphocytes; PMN = polymorphonuclear leukocytes; RBC = red blood cell; Eos = eosinophils; Hct = hematocrit; Hgb = hemoglobin; CFU = colony-forming unit; est = estimated.

TABLE 12. EFFECTS OF RFR *IN VIVO* ON THE IMMUNOLOGICAL SYSTEM*

Effects	Species	Exposure Condition				References
		Frequency (GHz)	Integsity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Increase in lymphoblasts in lymph nodes and increased response to SRBC	Mouse	2.95 (PW)	5	0.5 (est)	42 X 120	24
Increase in "spontaneous" lymphoblast transformation of cultured lymphocytes	Rabbit	2.95 (PW)	50	0.8 (est)	24-48 X 120	24
Increase in lymphoblasts in spleen and lymphoid tissue	Mouse	3.105 (PW)	20	2 (est)	6-8700	205
Increased transformation of unstimulated cultured lymphocytes and decreased mitosis in PHA-stimulated lymphocyte cultures	Chinese hamster	2.45 (CW)	50, 150, 300 or 450	2.3, 6.9, 13.8, or 20.7	5 X 15	61
Transient decrease and increased response of cultured lymphocytes to PHA, Con A, and LPS	Mouse	2.45 (CW)	50 or 150	3.6 or 10	1-17 X 30	203
Increased mitosis of PHA-stimulated lymphocytes	Rhesus monkey	0.01-0.027 (PW)	13,200	0.4-2.0 (est)	1 X 30	206
Increase in CR^+, FcR^+, and Ig^+ spleen cells. Increased response to B-cell mitogens. Decrease in primary response to SRBC	Mouse	2.45 (CW)	--	14	1 or 3 X 30	14,207,208
Increase in CR^+ and FcR^+ spleen cells	Mouse	2.45 (CW)	--	11.8 5	1 X 15 1 X 30	209
Increase in CR^+ spleen cells, strain specificity	Mouse	2.45 (CW)	--	10-19	1 X 30	10
Increase in CR^+ spleen cells	Mouse	2.45 (CW)	400	28	1 X 30	17
Increased lethality of endotoxin	Mouse	2.45 (CW)	200, 300	12, 18	1 X 120	210
Increase in response of cultured lymphocytes to T- and B-cell mitogens (Perinatal exposure)	Rat	2.45 (CW) 0.425 (CW)	50 100	1-5 3-7	57 X 240 47 X 240	99,201
No change	Mouse	2.45 (CW)	50-350	4-25	1-22 X 15 or 30	13
No change	Rat	0.1 (CW)	460	2-3	57 X 240	211
Increase in T and B lymphocytes in spleen. Decrease in DTH	Mouse	0.026 (CW)	8000	5.6 (est)	1 X 15 or 10 X 15	18
Reduction of lymphocyte traffic from lung to spleen	Mouse	2.6 (CW)	50 or 250	3.8 or 19	1 X 60	19
Decrease in NK activity, increase in macrophage phagocytosis	Mouse	2.45 (CW)	300	21	2 or 9 X 90	212
Decrease in NK activity	Hamster	2.45 (CW)	250	13	1 X 60	213
Increase in macrophage viridicidal capacity	Hamster	2.45 (CW)	250	13	1 X 60	214
Decreased response to PWM	Rabbit	2.45 (CW)	100	1.5	180 X 1380	196
No change	Quail	2.45 (CW)	50	4.03	12 X 1440	215
Decrease in tumor development	Mouse	2.45 (CW)	-- (near-field application)	35	11-14th day of gestation or 11-14 & 19-45 X 20	216
Decreased granulocytic response	Rabbit	3 (CW)	30	0.5	42-84 X 360	217
Tumor regression and increase in antitumor antibodies and anti-BSA	Rabbit	1.356	(near-field application)	(local hyperthermia)	1 X 10-15	218
Tumor inhibition and immune stimulation	Rat	2.45 (CW)	(200W)	(local hyperthermia)	3-6 X 45	219
Increased tumoricidal activity in lymphocytes and macrophages	Mouse	1.356	6000-9000	(local hyperthermia)	1 X 5	220
Tumor regression	Mouse	3 (CW)	400	28 (est)	1-14 X 120	221
Increase in lung cancer colonies and inhibition of contact sensitivity to oxazolone	Mouse	2.45 (CW)	500	36 (est)	4, 7, 10 or 14 X 120	222
Decrease in response to BSA	Rabbit	1.356	(near-field application)	(local hyperthermia)	3 X 60	223

*Adapted from EPA (1). See text for discussion of selected studies. SRBC = sheep red blood cells; PHA = phytohemagglutinin; Con A = concanavalin A; LPS = lipopolysaccharide; CR^+ = complement-receptor positive; Ig^+ = immunoglobulin positive; FcR^+ = receptor for Fc portion of immunoglobulin; DTH = delayed-type hypersensitivity; PWM = pokeweed mitogen; NK = natural killer cell; BSA = bovine serum albumin; CFU = colony forming unit; est = estimated; CW = continuous wave; PW = pulsed wave.

TABLE 13. EFFECTS OF RFR *IN VITRO* ON THE IMMUNOLOGICAL SYSTEM*

Effects or Endpoint	Species	Exposure Conditions				References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration (days X min)	
Increased blastogenesis of exposed lymphocytes *in vitro*	Human	3 (PW)	70 140		3-5 X 240 3-5 X 15	22
Increased blastogenesis of lymphocytes	Human	10	50-150		Observed effect only when culture temperature approached 38°C	4
No effects on mononuclear leukocyte viability, DNA, RNA, total protein, interferon synthesis	Human	2.45 (CW)	--	0.5-4	1 X 120	26
No effects on mononuclear leukocyte viability, DNA, total protein, synthesis	Human	2.45 (PW)	--	0.29-4	1 X 120	28
No change in spleen cell response to PHA, Con A nor LPS	Mouse	2.45 (CW)	100	19	1 X 60, 120, or 240	224
No change in blood lymphocyte response to PHA	Rat	2.45 (CW)	50, 100 or 200	0.7, 1.4 or 2.8	1 X 240, 1440, or 2640	225
No change in viability or growth of lymphoblast cell lines (Daudi and HSB_2)	Human	2.45 (CW)	100-5000	25-1200	1 X 15	226
Decreased macrophage phagocytosis	Mouse	2.45 (CW)	500	(15 J/min)	1 X 30	227
Liberation of granulocyte intracellular hydrolytic enzymes and increased death	Rabbit	3 (CW)	10 or 50		1 X 15, 30, or 60	228

*Adapted from EPA (1). See text for discussion of selected studies. PHA = phytohemagglutinin; ConA = concanavalin A; LPS = lipopolysaccharide; PW = pulsed wave; CW = continuous wave.

TABLE 14. EFFECTS OF RFR ON CLINICAL CHEMISTRY AND METABOLISM*

Effects	Species	Exposure Conditions				Δt (°C)	References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration (days X min)		
No effect on serum chemistry values	Rat	0.918 (CW)	25	1.0	91 X 600	0	157
Increase in serum glucose	Rabbit	2.45 (CW and PW)	50, 100, 250	0.8-4.0 (est)	1 X 120	0, 0, 1.7 (PW) 0, 0, 2.9 (PW)	229
Increase in blood urea nitrogen	Rabbit	2.45 (CW)	250	4.0 (est)	1 X 120	2.9	
No increase in blood urea nitrogen	Rabbit Rabbit	2.45 (CW) 2.45 (PW)	50 and 100 50, 100, 250	0.8, 1.6 (est) 0.8-4.0 (est)	1 X 120 1 X 120	0 0, 0, 1.7	
Increase in uric acid values	Rabbit	2.45 (CW and PW)	100, 250 (Neg 50)	1.6, 4.0 (est) (Neg 0.8)	1 X 120 1 X 120	0, 0, 1.7 (PW) 0, 0, 2.9 (CW) 0	
No effect on other serum chemistry values	Rabbit	2.45 (CW and PW)	50, 100, 250	0.8-4.0 (est)	1 X 120	0, 0, 1.7 (PW) 0, 0, 2.9 (CW)	
Increased iron and manganese levels in brain	Rat	1.6 (CW)	800	48 (est)	1 X 10	4.5	230
Decrease in specific metabolic rate (Ambient Temp = 24°C)	Mouse	2.45 (CW)		10.4 (Neg 5.5)	1 X 30		231
Increase in specific metabolic rate (Ambient Temp = 35°C)	Mouse	2.45 (CW)		8.6 (Neg 3.6)	1 X 30	0	232
Increased NADH fluorescence; Decreased ATP; Decreased CP; (Exposed brain)	Rat	0.591 (CW)	138	0.36-2.2	1 X 0.5	0	233
Decreased ATP; Decreased CP; (Exposed brain)	Rat	0.591 (CW)	50	0.13-0.8	1 X 0.5	0	233
Increase in oxygen consumption	Rat	2.45 (120Hz PW)		6.5, 11.1 (Neg 4.5)	1 X 30	0.9, 1.8, 0.4	117
Decrease in metabolic heat production	Monkey	2.45 (CW)	60	0.9	1 X 10	0	234
No effect on blood coagulation (Exposed plasma)	Human	2.45 (CW)	100-2800	1.3-38 (est)	1 X 30	Not reported	235

*Adapted from EPA (1). Neg = effect not found at value indicated; CW = continuous wave; PW = pulsed wave; est = estimated; Δt = Colonic temperature increase.

TABLE 15. NEUROENDOCRINE RESPONSES TO RFR EXPOSURE*

Effects	Species	Exposure Conditions				Δ(°C)	References
		Frequency (GHz)	Intensity (W/m^2)	SAR (W/kg)	Duration days X min		
Increased thyroxine and triiodothyronine	Dog	2.45 (CW)	720-2360	58-190	1 X 120	2-8 (thyroid temp.)	235, 237
No effect on thyroid gland nor thyroid hormone	Rat	2.45 (CW)	10, 100, 1000 10,100	0.25-25 0.25-2.5	1 X 10-45 56 X 480	$\leq$100 W/m^2=0 1000 W/m2 $\geq$5	238
No effect on thyroid function	Rat	2.45 (CW)	100, 200, 250	2, 5, 6.5 (est)	1 X 240-960	$\leq$200 W/m^2 = 0 250 W/m^2 = 1.7	239
Decrease in serum protein-bound iodide, thyroxine and thyroxine/serum ratio	Rat	2.45 (CW)	150	3.8 (est)	2.5 X 1440	0	239
Increase in thyroid hormone	Rabbit	3 (PW)	50	0.25-0.75 (est)	48 X 180	Not reported	240
Decrease in serum thyroxine levels	Rat	2.45 (CW)	200 (Neg 10-100)	5 0.25-25	1 X 240-480 1 X 60-480	0-0.6 0.6-1.4	241
Increase in corticosterone levels	Rat	2.45 (AM, 120 Hz)	400-700 (Neg 10-200)	8.4-14 (Neg 0.21-4.2)	1 X 60	1.3-3.0 0-0.6	242
Decrease in thyrotropin levels			100, 400-700 (Neg 10-50, 200)	2.1-14.7 (Neg 0.21-4.2)		0-3.0 0-0.6	
Increase in corticosterone levels			100-400 (Neg 10-50)	2.1-8.4 (Neg 0.2-1.0)	1 X 240	0.3-2.1 0	
Decrease in thyrotropin levels			250, 400 (Neg 10-200)	0.6-2.1 0-1			
No effect on thyroid, pituitary, or adrenal gland weights nor growth hormone levels	Rat	2.45 (CW)	10-200	0.25-25	1 X 60-480	0-1.4	241
No effect on thyroid, anterior pituitary gland, adrenal, prostate or testes weights; no change in follicle-stimulating hormone nor gonadotropic hormone levels	Rat	2.86-2.88 (CW)	100	1-2 (est)	36 X 360	Not reported	243
Increase in leutinizing hormone	Rat	2.86-2.88 (CW)	100	1-2 (est)	36 X 360	Not reported	243
Increased adrenal weights and significant adrenal response (infants exposed)	Rat	2.45 (CW)	400	20-60 (est)	6 X 5	1.5-2.5	111
Increased plasma corticosterone levels	Rat	2.45 (CW)	500, 600 (Neg 130-400)	11.5, 13.8 (est) (Neg 3.0-9.2)	1 X 30-60	130 W/m^2=0.5 200 W/m^2=0.7 300 W/m^2=0.9-1.4	244
			200-400 (Neg 130)	4.6-9.2 (est) (Neg 13, est)	1 X 120	400 W/m^2=1.3-1.4 500 W/m^2=1.6-2.4 600 W/m^2=2.5-2.9	
Increase in corticosterone	Rat	2.45 (AM, 120 Hz)	500,600	8.3, 9.6	1 X 60	Not reported	245
No effect on serum corticosterone levels	Rat	0.918 (CW)	25	1.0	91 X 600	0	157

*Adapted from EPA (1). CW = continuous wave; PW = pulsed wave; AM = amplitude modulated; est = estimated; Neg = effect not found at value indicated;

Δ(°C)= change in colonic temperature.

THE CUMULATIVE EFFECTS OF LONG-TERM EXPOSURE TO LOW LEVELS OF RADIOFREQUENCY RADIATION (RFR)

by

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SUMMARY

One of the primary concerns for the effects of nonionizing radiofrequency radiation (RFR) has been accumulation of subtle injury over a long period of time, resulting in a delayed expression of deleterious effect. Of the more than 6,000 articles in the literature today, the vast majority involve acute exposures at levels where significant thermal energy was deposited. The resulting effects, in most cases, could be explained on the basis of the specific energy absorption, expressed as watts per kilogram (W/kg), with a generally accepted threshold for effects of 4 W/kg. The advocacy of nonthermal mechanisms by means of mathematical modeling, theoretical predictions, and in vitro studies, raised the possibility of subtle injury or alteration in function which, over time, would be expressed as a harmful bioeffect. The few studies addressed to this problem suffered one to several deficiencies in method, including relatively short duration of exposure, small numbers of subjects, inappropriate end-points, or incomplete dosimetry. Epidemiological appraisals also were not adequately performed or could not clearly relate findings to exposure history. A recently completed project, performed by personnel at the Bioelectromagnetics Laboratory, University of Washington School of Medicine, Seattle, Washington, and funded by the U.S. Air Force School of Aerospace Medicine, was directed toward these concerns. Over a 4-year period of planning, pilot study, and definitive experiment, a lifetime exposure was given to a population of test animals (100) whose state of health, growth, and cause of death were closely monitored. An equal number of sham-exposed animals served as a comparison population. After 25 months of exposure, and at the point where 90% mortality in both groups had occurred, the remaining subjects were sacrificed and assayed. The methods and results have been published as a series of nine technical reports. The overall conclusion was that no cumulative ill effects could be attributed to the life-long exposure at absorption rates of 0.4 W/kg or less.

INTRODUCTION

One of the primary concerns for the effects of nonionizing radiofrequency radiation (RFR) has been accumulation of subtle injury over a long period of time, resulting in a delayed expression of harmful effect. Over the past 15 years, the expression of this concern has become more vocal and various official bodies have identified long-term, low-level effects as a main research priority (1,2). The effect of this unresolved issue has been considerable on those entities of government and industry which operate RFR emitters (3,4). By necessity, transmission exposes both operational personnel (occupational) and members of the general public (environmental). This second aspect of the exposure condition effectively removes the responsibility for ensuring the well-being of the exposed population from the entity producing the exposure to other government agencies. The philosophical rationale for the exposure and the ingredients of the risk-benefit equation also change. These factors combine to affect the siting and mode of operation of any given emitter through their effect on standards and permissible exposure limits. The larger the degree of uncertainty, the more inclined standard-setters are toward extreme conservatism since the level of risk cannot be even roughly bounded. This is particularly true if one applies the reasoning underlying ionizing radiation permissible exposure levels (PELs), that any level of exposure produces some injury, i.e. a linear extrapolation to zero.

In the case of RFR, a general consensus has been achieved among standard-setters that, for acute exposures, the threshold for ill effects lies above a specific absorption rate (SAR) of 4 W/kg (5,6,7,8). Since hazard, i.e., injury, has only been verified above this threshold, one might conclude that the current approach of applying a safety factor of 10 or more to this level would adequately protect the population at risk. Unfortunately, this is not the case. Lack of credible data derived from true long-term experiments with animals or from epidemiological studies of defined exposures to a human population has prevented a consensus from being reached for these kinds of exposures. Literature reports of long-term animal studies are quite diverse, with the definition "long-term" or "chronic" being applied to experiments involving exposures for only a few hours over a time course of days or weeks. Before 1977, the reports were also hampered by inaccurate or missing details of the dosimetry methods employed. Lack of a common denominator, such as the SAR, made comparisons among experiments from different laboratories difficult. Endpoints measured also varied widely, with the selection usually determined by the investigator's field of training or interest, or by using those tests most readily performed. This lack of specificity produced conflicting and unrelated data sets, e.g., a few hematological parameters, a

behavioral task, an immunological test. I will briefly review the state of knowledge regarding epidemiological assessments to detect RFR effects and those animal studies which relate to possible cumulative effects.

EPIDEMIOLOGY

The most directly applicable experimental evidence relative to possible bioeffects of exposure to RFR from any specific RFR-emitting system would be from studies in which humans were exposed to the frequencies and waveform characteristics of that kind of system, for appropriate durations, at the maximum average power densities likely to be encountered.

Existing epidemiologic studies, though extensive and reasonably well done, are subject to inherent defects such as imprecise classification of the individuals with regard to RFR exposure and unavailability of complete sets of medical reports, death certificates, or health questionnaires.

Epidemiology, as used here, refers to studies of whether one or more health-related conditions can be associated statistically with purported or actual exposure of humans to RFR (in contrast with assessments based on extrapolation from data on animals to humans). Epidemiologic results tend to be based on imprecise estimates of exposure characteristics (frequency, power density, and duration), and in some cases no quantitative estimates at all. The extent to which the control group matches the exposed group is sometimes open to question. Because matching of all relevant factors except exposure is the basis of concluding that any observed differences between groups are related to the RFR exposure, selection of an appropriate control group is critical. Despite these limitations, such studies do provide almost the only information available on possible effects of actual RFR exposure in humans.

Ocular Effects

One of the early concerns was ocular damage from RFR exposure. Various cases of individuals with cataracts ascribed to RFR exposure have been reported from time to time. Indeed, it is likely that in some of these cases, occupational exposure to high levels of RFR had resulted in frank thermal damage to the eye. Not clear, however, was whether chronic exposures to low levels of RFR could be cataractogenic. The epidemiologic studies referenced are representative and were inspired by reported cases of ocular damage at high levels (9,10,11,12). The consensus was that exposures at low levels, 100 mW/cm^2, (below the levels at which the internal ocular temperature is elevated above 41°C (13) do not predispose to cataracts at a later time.

As in other epidemiologic studies, detailed exposure histories (frequencies, intensities, durations, and so on) could not be determined with accuracy, if at all, for either the exposed or the control groups in these ocular studies. However, the exposed groups quite likely did receive more RFR exposure. It is noteworthy that interest in RFR ocular effects have waned in recent years, as evidenced by the relatively small research effort devoted to it. Two recent studies have made preliminary reports of corneal damage at low levels, but to date, the results have not been published nor the experiments replicated.

Congenital Defects

Two reports were published claiming RFR effects on fetal development based on retrospective epidemiology (14,15). Detailed examination of the data by others failed to support any causal relationship with RFR exposure (16,17).

General Health Effects

Many attempts have been made to evaluate the health of workers assumed to be occupationally exposed to RFR (18-27).

In summary, none of these U.S., Polish, and Czechoslovakian epidemiologic studies offers clear evidence of detrimental effects associated with exposure of the general population or of selected occupational groups to RFR. The Soviet findings, which are consistent with the voluminous early Soviet literature, suggest that occupational exposure to RFR at average power densities less than 1 mW/cm^2 does result in various symptoms, particularly those associated with disorders of the central nervous system (CNS). Because the USSR symptomatology has not been reported in western studies and because of the marked differences in their procedures used for reporting data, it is difficult to accept the USSR epidemiologic studies at face value.

The U.S. Embassy in Moscow was subjected to RFR exposure from 1953 until February 1977 (28). Within rooms having the highest RFR levels, i.e., those with windows and doors in outside walls toward the irradiation sources, the average power densities were typically about 0.104 mW/cm2 within 60 cm of a door or window, and 0.0025 mW/cm^2 elsewhere in the room. The highest power density reported was 0.024 mW/cm^2, which occurred in one room during a 2-h period of unusual signal strength on 24 January 1976 (29). Lilienfeld, et al, (30) compared the health of US personnel assigned to the Moscow embassy from 1953 to 1976 with the health of those assigned to other U.S. Eastern European embassies. The investigators noted several limitations of their study but were able to conclude that no differences were discerned between the Moscow and control groups in total mortality or mortality from specific causes, nor between dependent children or adults of the Moscow and control groups.

MUTAGENESIS, CARCINOGENESIS, AND CYTOGENETIC EFFECTS

One frequently expressed concern about RFR is that it may be mutagenic or cause cancer. As suggested by Ames (31), mutagenesis and carcinogenesis are believed to be related, and indeed many chemicals are screened for potential carcinogenicity with bacterial mutation tests.

Several studies for mutagenic effects of RFR at various frequencies, power densities, and durations have been done on bacteria and yeasts (32-34). No mutagenic effects attributable to RFR exposure were reported.

Four studies for mutagenic effects of RFR in fruit flies also yielded negative results (35-38), two studies evaluated abnormalities in chromosomes in RFR exposed cells (39-40) and two studies looked for effects on sister chromatid exchanges (41-42). Most of these studies were of relatively short duration of exposure, some incident power levels were quite high. Where effects were seen, thermal events could not be discounted.

A study by Prausnitz and Susskind (43) implied an association between RFR exposure and cancer incidence. They exposed male mice to pulsed 9.27-GHz RFR at 100 mW/cm^2 average power density for 4.5 min per day, 5 days per week, for 59 weeks. Leukosis was present among both groups, with a higher incidence for the exposed mice. Leukosis was described in the paper as "cancer of the white blood cells." However, the authors apparently confused leukosis with leukemia or cancer of the circulatory system.

In addition, two other factors must be considered. First, the incidence of leukosis was greater in the exposed mice, but their survival was also greater. This would be considered unusual for most forms of mouse leukemia. Second, in the exposed mice, the incidence of leukosis was greater during but not following exposure. This would imply that spontaneous remission of the "cancer" occurred after cessation of exposure. For true cancer, this would be considered quite improbable. Overall, the data did not provide any valid evidence that chronic RFR exposure induced any form of cancer in the exposed mice.

In summary, there is no evidence that exposure to RFR induces mutations in bacteria, yeasts, or fruit flies. The results of two studies indicated that RFR induces mutations in mammals; critical review has cast doubt on these findings. Other studies have shown no mutagenic effects of RFR on mammals; evidence for cytogenetic effects is mixed. The lowest power density at which such effects were reported was 20 mW/cm^2; however, Chen (39) failed to find cytogenetic effects at 200-500 mW/cm^2. Last, there is no credible evidence that chronic exposure to RFR induces any form of cancer in animals, even at power densities as high as 100 mW/cm^2.

TERATOGENESIS AND DEVELOPMENTAL ABNORMALITIES

Teratogenesis in mammals is the production of physical defects in conceptuses that affect their in-utero development. The term "developmental abnormalities" as used here refers to processes affecting the development of infants after birth. Teratogenic and developmental abnormalities occur naturally at a low rate in most animal species, and relatively little is known about their cause. In a few cases, however, specific agents have been shown to cause significant teratogenic effects; hence, the possibility of teratogenic effects from RFR is an appropriate matter of public concern.

Numerous teratogenic studies involving RFR have been reported utilizing animal models ranging from beetle pupae to monkeys, with frequencies and power densities differing greatly from study to study (44-58).

In the studies showing demonstrable teratogenic effects of exposure to RFR, the power densities used produced SARs that were capable of generating significant heat loads in the animals. In general, the results indicate that a threshold of heat induction or core-temperature increase must be exceeded before teratogenic effects are produced and that RFR per se is not teratogenic. More recent studies (59,60) using pregnant ewes have shown no potential for uterine temperature increases in large mammals exposed to RFR levels likely to be encountered by pregnant workers.

OTHER LONG-TERM STUDIES

Effects from chronic exposure have been reported as far back as the 1930's (61,62). Another study involving chronic exposure (63), exposed mice to 800-MHz RFR at 43 mW/cm^2 for 2 h per day, 5 days per week, for 35 weeks. Some deaths occurred during exposure and were attributed to thermal effects caused by faulty positioning of the animal holders. The mean life span of the remaining exposed mice did not differ significantly from that of the controls, general indications of health were the same in the two groups, and the incidence of cancer was the same in the exposed and control mice.

Baum et al (64) exposed rats to EMP (electromagnetic pulses) at a rate of 5 per sec continuously for 94 weeks. The spectrum of the EMP corresponded to an RFR center frequency of 450 MHz, and each pulse had an intensity of 447 kV/m. The exposures had no effect on blood chemistry, blood count, bone marrow cellularity, fertility, embryo development, cytology, histology, or cancer incidence.

Numerous other studies in the literature exposed small rodents to frequencies from 918 MHz to 3 GHz at average power densities from 1.0 mW/cm^2 to 25 mW/cm^2. Biological endpoints were quite diverse and measured various elements such as hematology, histopathology, growth and development, blood chemistry, behavior, and endocrinology (65-71). Similar reports were generated in studies with rabbits, dogs, and primates (72-77). Reference should also be made to chronic environmental and ecological studies that have been reported (78).

While the above studies reported essentially negative results, there are many chronic studies which report bioeffects at very low levels of RFR (79-84). While not an exhaustive list, the cited papers are a representative cross-section. In considering this data, one must recognize the absolute essentiality of carefully handled, identically treated control animals.

Secondly, it is a fact that there are wide ranges of "normal" values for every species, and thermoregulatory mechanisms involve numerous neurophysiological events and reactions that do not constitute hazard or injury, but are normal thermal adaptation. Biologic effects do not necessarily constitute a biologic hazard.

Lastly, it should be clearly understood that a few milliwatts/cm^2 exposure at gigahertz frequencies is a thermal insult to small rodents, and hence equivalent hot air or hot water sham-irradiated control animals should also be included in a rigorous scientific study.

In summary, those studies reporting positive effects at very low power densities (<4 mW/cm^2) contain much incomplete as well as conflicting information. Replication of selected results in other (Western) laboratories has not been successful. Careful reading would suggest a lack of precision in dosimetry, and the reporting of periodic observations of cyclic phenomena as real differences. On the other hand, power densities of 5 mW/cm^2 and greater, at or near resonant frequencies for small animals, are thermally significant, and can initiate a wide range of biological events.

THE UNIVERSITY OF WASHINGTON LONG-TERM LOW-LEVEL STUDY

It was against this backdrop that the U.S. Air Force, in 1978, embarked on the most ambitious long-term low-level RFR study ever attempted. The study was conceived as a controlled study of the state of health of a test population large enough to yield statistically valid data, an identically treated population, except for the exposure, was also provided for, 100 animals in each group, or a total study population of 200.

Following two years of facility and equipment design, exposure device construction, protocol development and refinement, and pilot operation, a definitive study was initiated in September 1980. After 25 months of continuous exposure, the test was completed in September 1982. Data analysis and tissue examination were completed one year later, and the results have been published in a series of nine technical reports, of which the ninth is an overall summary and interpretation (85-93).

FACILITIES AND PROCEDURES

Exposure Criteria.

The first decision made was to select a test animal and exposure situation to best model the exposure of humans to 450 MHz. The selection of 450 MHz as the frequency of interest was based on two main facts. Public concern had been expressed over the operation of a phased array radar (PAVE PAWS), which operates with frequencies in this region. Also, this frequency produces, in man, a whole-body exposure which is reasonably well distributed. When scaled to the rat, this equates to a frequency of 2450 MHz. This resulted in the selection of a circular waveguide system and the Sprague-Dawley rat for the experimental system. This allowed the exposure of a large number of animals to a common RFR source while independently maintaining relatively constant and quantifiable electromagnetic coupling to each animal, regardless of location, posture, or movements. It was then necessary to choose a modulation frequency and the pulse parameters. Both selections were to some degree arbitrary, but were based on the same concept used to select the basic 2450 MHz exposure frequency, namely to simulate in the rat the same distribution and absorption pattern predicted in humans from exposure to a typical mid-range Air Force system. The modulation pattern chosen, 8 Hz, represented the dominant electroencephalogram (EEG) frequency of rats (94), and was included to encompass a reported effect of RFR exposure in vitro (95,96). These variables were arbitrarily fixed to limit the magnitude of the project.

The exposure system was characterized by exhaustive dosimetry, not only to ascertain the true pattern of energy absorption and distribution in the exposed subject, but also to maximize the extrapolation of the findings by scaling the exposure frequency and intensity to represent human exposure to comparable conditions.

Biological Assessment.

Considerable effort was made to select as endpoints not only biological effects previously reported from low-level microwave exposure (e.g., alterations of hematopoetic, immunologic, and specific blood chemistry indices), but also to look for possible cumulative effects on general health, metabolism, or lifespan. Only those endpoints were included which could be assessed without seriously compromising the health of the animal, the value of concurrent measurements, or the power of the statistical evaluations of the chosen endpoints. Peer review by researchers within the community concerned with the bioeffects of microwaves as well as the scientific community at large, tempered the final protocol.

The endpoints selected fall within 5 general areas; (1) behavior, (2) immunology, (3) hematology and serum chemistry, (4) growth and metabolism, and (5) longevity and cause of death. Although treated separately for the purpose of facilitating timely publication, the results are all interrelated and to treat them as isolated experiments is incorrect. Volume 9 (93) evaluates the overall study and makes the proper cross-correlations among endpoints.

Behavior

The sole behavioral endpoint included as part of the overall protocol for the long-term study was an assessment of open-field activity. It was chosen after a variety of suggested behavioral tests--including shuttlebox avoidance, activity wheel, discriminated T-maze, and various schedule-reinforced, bar-pressing paradigms--had been evaluated according to the following criteria.

First, the behavioral test selected should not jeopardize the health of the animals and thus interfere with the primary goal of the project, i.e., evaluation of the status of health throughout life, and effects on mortality. Second, the test should not lead to obvious reactions to stress or differential experience (e.g., shock density) based on level of performance during testing. Third, the test must be easily performed within the confines of the specific pathogen free (SPF) facility and the time schedule of the daily maintenance procedures. Fourth, the test must not be subject to bias on the part of the experimenter. Finally, the test must have a history of reported sensitivity in microwave-exposure studies.

The open-field test is not the most impressive of the behavioral test procedures considered, but it satisfies the selection criteria. It is simple in nature, does not rely on elaborate or time-consuming training procedures or shock-motivated performance, and can be routinely administered by laboratory personnel under the rigors of SPF control. East European researchers have used the open-field test extensively in studies of the bioeffects of microwaves. Their reports claim that it is the most sensitive of the behavioral tests used, revealing complex relationships between observed behaviors, length of exposure, and power density (97,98).

Assuming behavioral changes could be stress-related, corticosterone was periodically assessed for two purposes: (1) to monitor the general environment of the experimental animals and the handling procedures required by the daily maintenance schedule; and (2) to test specifically for corticosterone changes resulting from microwave exposure (99).

During the first year of the project five corticosterone sampling sessions were completed, at 12-week intervals, coincident with every other regularly scheduled bleeding session. During the second year, corticosterone assays were made only twice, 6 weeks before and at the time that the last surviving animals in each group were killed.

Except for corticosterone values in the first assessment period, open-field activity and corticosterone levels were not significantly altered by 2 years of exposure to low-level pulsed-microwave radiation. A statistically significant elevation of serum corticosterone was present in the first set of samples. This could have been due to random fluctuation, or the animals may have detected the exposure. In subsequent samplings there were no differences.

Immunology

Various studies have been published concerning the immunological responses of various experimental animals to microwave irradiation. These studies provide a basic framework for inquiry but, when viewed as a whole, reveal inconsistencies and inadequacies that stimulate controversy concerning the significance of basic findings with regard to potential hazard to human health. Stimulatory effects indicated by the results of some studies have not been reproduced in others.

To examine this area of interest, the following immunological tests were performed coincident with the interim killing of 10 animals from each treatment group after 12 months of exposure: response of splenic lymphocytes to various mitogens, plaque-forming ability, complement-receptor formation, and enumeration of B- and

T-cells. The same immunological tests were also performed on 10 animals from each group coincident with the terminal kill of the 24 animals remaining after 25 months of exposure.

After 13 months of RFR exposure, the exposed experimental animals had a significant increase in both splenic B- and T-cells when compared with the sham-exposed group. This apparent general stimulation of the lymphoid system in the RFR-exposed animals was not detected in the animals evaluated after 25 months of RFR exposure. Comparison of the exposed and sham-exposed rats in the terminal kills did not reveal any significant differences in the percentage or total numbers of B- and T-cells per spleen. The lack of a significant difference in the terminal-kill animals may be the result of age and the onset of immunosenesence, or it may reflect a random difference at the 13-month period, possibly due to the small number of animals examined.

No significant differences were seen between the exposed and sham-exposed rats in the percentage of complement-receptor-positive cells in the spleen at either time of examination, interim or terminal. This indicates no difference between the treatment groups in the maturation of lymphocytes as indicated by this procedure.

The plaque assay performed on animals immunized with sheep red blood cells (SRBC) indicated no statistically significant alteration of the reticuloendothelial system, which first processes the SRBC antigen, and no deficiency in the B-cells' ability to produce antibodies in the presence of T-cells, as the SRBC antigen is T-cell dependent.

The mitogen-stimulation studies following 13 months of exposure revealed a significant difference between groups in their responses to various B- and T-cell specific mitogens. A nonsignificant increase in response to phytohemagglutin (PHA) and a significant increase in response to lipopolysaccharide (LPS) and pokeweed mitogen (PWM) was detected in the RFR-exposed animals. The exposed animals also had a significantly increased response to concanavalin (ConA) and a decreased response to purified protein derivative (PPD) ($\underline{p}$ = .01) as compared to the sham-exposed animals. Mitogen response data were not available from the 25-month exposure studies because the lymphocyte cultures failed to grow.

The equivocal and, in some instances, incomplete nature of these data resulted in a follow-on immunological study being initiated, utilizing a larger number of animals, more precise stimulation assays, and more sophisticated, computer controlled, cell sorting and counting methods.

Preliminary data from the animals examined following six months of exposure do not confirm any previous findings, although, not surprisingly, a few of the 26 parameters examined exhibited differences between control and exposed groups

The numbers of hematopoietic progenitor cells were quantitated in the femurs of exposed (experimental) and sham-exposed (control) rats. The data demonstrated that exposed rats had significantly elevated total numbers of colonies (p, 0.014); of macrophage colonies (p, 0.046); and of granulocyte colonies (p, 0.016). No differences were observed in the numbers of mixed colonies.

In order to detect functional alterations induced by RFR irradiation in subpopulations of lymphoid cells from the spleen and thymus, single cell suspensions of each organ were cultured in vitro with the optimal concentration of mitogens and assayed for DNA synthesis on the peak day of DNA synthesis response. No significant differences between exposed and sham-exposed rats were observed in the proliferation of thymocytes to ConA, PHA, and PWM. This result was unchanged when rats with outlying values were excluded from the analysis.

The DNA synthesis response of splenocytes to a panel of mitogens was evaluated us ng the observed scale and on the natural logarithmic (ln) scale. A marginally significant difference was observed in the PPD response (ln) scale), which is a B cell specific response. In this case, exposed rats manifested a higher DNA synthesis response than did sham-exposed (control) rats., No other significant group differences were observed for the other mitogens (p greater than 0.05).

Thymocyte suspensions were prepared from exposed and sham-exposed rats and each subsequently stained with FITC goat anti-rat IgG (B cells) and FITC anti-Thy 1.1 (T cells). The cells were analyzed on the Orthocytofluorograph. No significant differences between groups were observed in the percentage of B cells (s-Ig) or T cells (Thy 1.1) in the thymii. The population mean expression of s-Ig (B cells) and of Thy 1.1 cell surface antigen (T cells) did not differ significantly between exposed and sham-exposed animals.

The analysis of the marrow and spleen cells from exposed and sham-exposed rats for the presence of B cells and T cells revealed the following: (a) no evidence of group differences in the percentage of T and B cells and in the mean expression of s-Ig and Thy 1.1 or B or T cells, respectively, was observed; (b) marginally significantly group differences (p, 0.05) on the percentage of total viable B cells in the marrow was noted where the exposed animals had fewer B cells in the marrow; (c) no other statistically significant group differences were found.

The analysis of correlated light scatter parameters revealed that exposed animals had significantly fewer marrow cells with low light scatter characteristics than did sham-exposed rats. All the other parameters showed no group effects.

These data are continuing to be analyzed and no overall hypothesis for the effects found is readily apparent. The biological significance is likewise undetermined, and will likely remain so.

Hematology and Serum Chemistry

A rather formidable battery of hematological, biochemical and hormonal assays, as well as a complete serum protein analysis, were performed at periodic intervals throughout the study. Blood samples were obtained fortnightly during the first year, and at 12-week intervals thereafter.

The major conclusion that can be reached from the results of evaluations of the hematology, serum chemistry, protein electrophoretic patterns and fractions, and thyroxine levels is that any significant alterations of these parameters during the lifetime of the exposed animals were to be expected with age and were not due to exposure to pulsed microwave radiation.

The remaining two major categories of endpoints represent the most informative sets of data. Growth and metabolism reflect the total effect of the organism's expression of its genetic makeup in a closely controlled environment, making the comparison of the two conditions, exposed versus non-exposed, an accurate insight into any expression of cumulative treatment effects. The obverse of this reflection is represented by the data on mortality and cause of death. One can think of it as the subtracting away from the ideal life span, thereby accentuating any cumulative exposure effect which would either accelerate mortality over time, alter the predominant mode of death or selectively advance the time of appearance of disease entities associated with aging.

Growth and Development

Data for growth and development was gathered by daily measurements of food and water consumption, and increase in body weight. In addition, a small subsample of animals were examined at the interim kill, 13 months post-exposure, and on termination of the exposure 12 months later. A total body carcass analysis was done for total moisture, total ash, total crude fat, protein-bound nitrogen and non-protein bound nitrogen. Total crude fat was further analyzed for specific fatty acid content and for 27 mineral elements. Prior to the carcass analysis, vital organs had been dissected and weighed. From this preponderance of data, only one value showed significant treatment effect, adrenal weight. Detailed examination of the cellular structure, however, showed this difference to be due to adrenal tumors rather than an overall increase in functional adrenal tissue. Previously cited theories regarding a hyperplastic stress response are not supported by the findings. Growth curves for the two test populations were nearly identical (Figure 1).

The attempt to assess oxygen consumption as a metabolic indicator was less than fully successful. Data from young animals were highly variable and difficult to interpret. For the older animals, the data were more consistent and did not reveal significant treatment differences. A three-month follow-on experiment to reexamine these metabolic parameters in younger animals detected no significant treatment difference. A more ambitious metabolic experiment is now in progress in an attempt to establish exposure/environmental thresholds of response.

Longevity and Cause of Death

This was the first project to systematically evaluate the histopathology of lifetime exposure to low-level RFR. It was carefully designed to record the onset of lesions, to determine their nature, examine their association with natural aging and to detect any differences between control and experimental population in respect to these factors. Much attention was paid to experimental animal quality control. All animals were checked every night to detect any animals which died or were near death after regular working hours. This helped to limit postmortem autolysis of the tissues. Forty-four rats were killed as part of the interim and terminal evaluations.

One of the overall parameters evaluated was life expectancy. Cumulative survival curves were completed (Figure 2) and the effect of eaoh animal's death plotted. Mean survival time for the exposed animals was 666 days (± S.E. 15 days), for the sham-exposed mean survival was 643 days (± S.E. 18 days). By appropriate statistical tests, no significant effect existed in either early or late mortality (Figure 2).

Overall analysis of the major causes of death support the null hypothesis of no association between cause of death and treatment, i.e., exposure. Time of occurrence, early or late, was not different, except for an earlier onset of one condition, urinary obstruction, in the sham exposed.

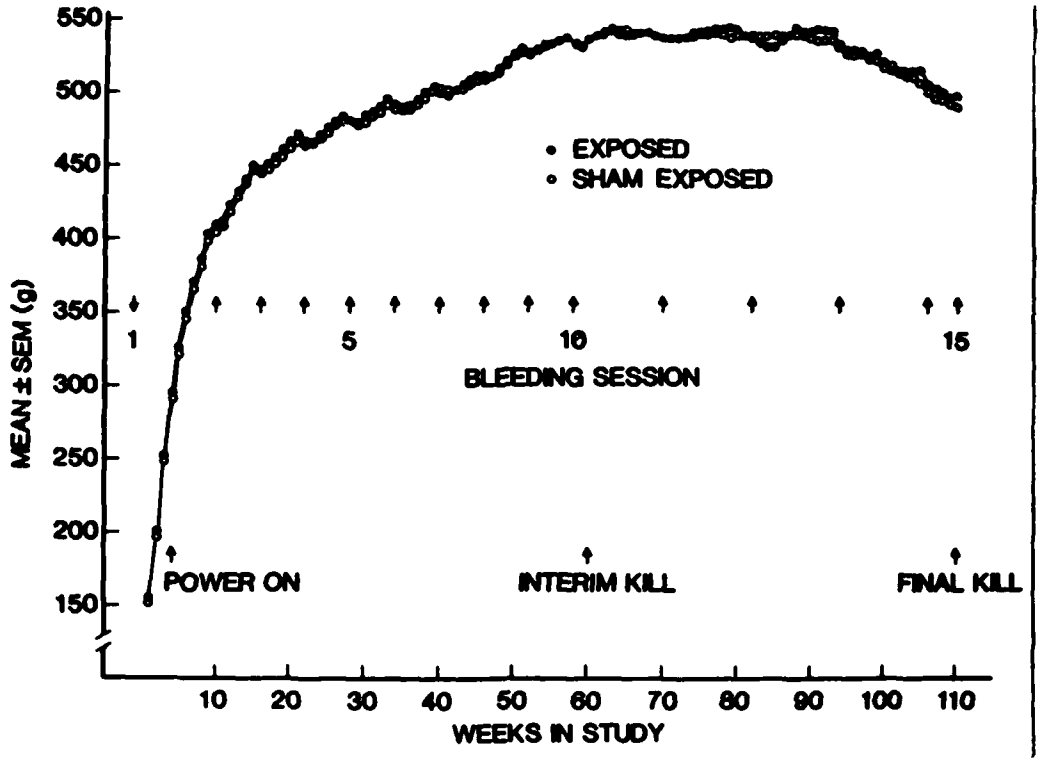

Figure 1. Weekly means for body mass during study. Fluctuations represent recurrent loss of body mass following each blood collection session.

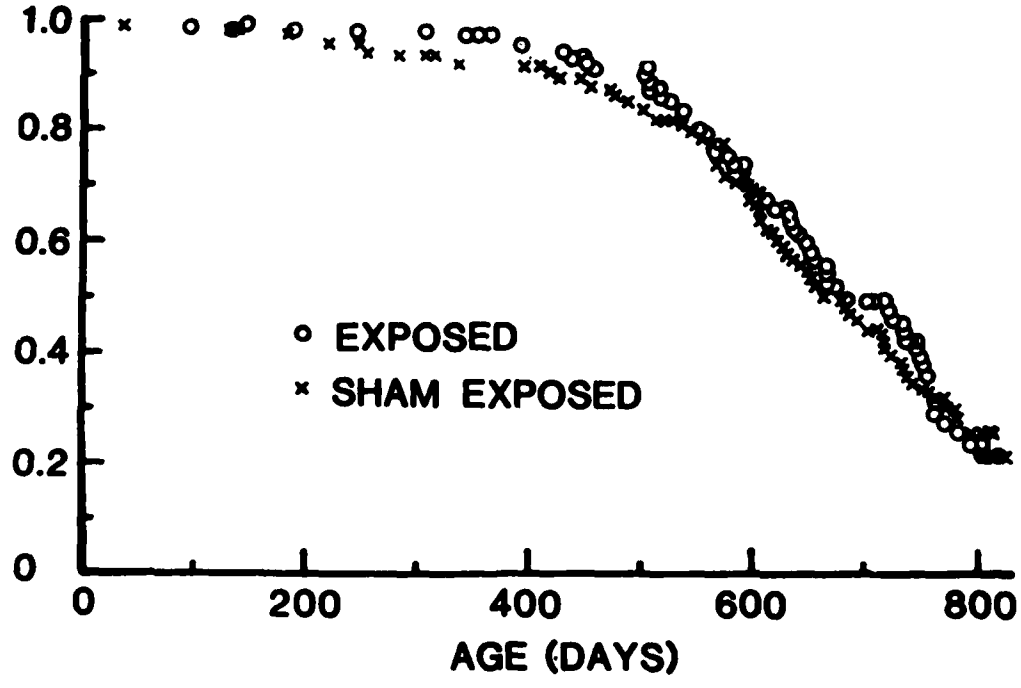

Figure 2. Cumulative mortality expressed as percent surviving by day of exposure.

The neoplastic lesions were identified as benign or malignant, with the latter further classified as primary or metastatic. The low incidence of neoplasia with no specific increase in any specific organ location or tissue type led to collapsing the data for statistical analysis, without regard to area of occurrence. Neither group had an excess of tumors when compared to each other, although the exposed rats had a statistically higher number of malignant neoplasms when compared to the sham exposed. The collapsing of sparse data without regard for tissue origin obscures the true biological activity. It is not generally accepted procedure to collapse tumor data in this manner, and in most studies of carcinogens, one would look for either a statistically significant increase in a particular type of tumor normally present or any incidence in the treatment group of tumors not usually seen in the species in question. Neither of these criteria were met in the RFR study (100). Distinctions between benign and malignant tumors are not usually emphasized as the initiation process is thought to be the same in both cases.

Some criticism was leveled during the early stages of the University of Washington study, challenging the concept of frequency scaling. That is, equating a 2450 MHz exposure in the rat to a 450-MHz exposure for man, even though this is a widely accepted practice, and was validated by a major dosimetry effort as part of the study. To address this issue, the Engineering Experiment Station at the Georgia Institute of Technology (GIT) was contracted to design, construct, and operate a multi-animal exposure facility in the 425-450 MHz region. This was done (101,102) and the facility is now being used to study the effects of prolonged exposure to 435 MHz RFR. Experiments are currently underway to evaluate the response of cardiovascular dynamics to exposure, utilizing rats with chronic aortic catheter implants, however results are not yet available.

Until solid human epidemiological data is available, some concern for long-term effects will remain. The difficulty in assessing implied very subtle effects without a distinguishing lesion or disease entity will continue to fertilize the blossoming of anecdotal epidemiology data. In the meantime, rational review of the existing data provides no reason to predict adverse consequences from lifespan exposure to currently allowable levels of RFR.

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The Medical Results of Human Exposures To Radio Frequency Radiation

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Summary

The United States Air Force has conducted a clearly defined and effective radio frequency radiation (RFR) protection program since 1970. As an important part of the program, since 1972 the Air Force Medical Service has investigated RFR exposure incidents involving more than 330 individuals; of which 58 were determined to have been actually overexposed with another 17 found to have been inconclusive. Medical evaluations of the exposees have been extensive and the findings almost universally unremarkable. This paper presents a short history of the program's evolution, some examples of exposure incidents and some general impressions of the clinical evaluations of the confirmed exposees.

Introduction and Background

The U.S. Air Force has always placed great emphasis on safe working conditions for its personnel. Radiation protection programs for the work force have enjoyed a high priority and an excellent reputation for effectiveness over the years.

Prior to 1970, radio frequency radiation (RFR) protection programs within the Air Force community were largely managed at base-level. In mid-1970, the USAF Radiological Health Laboratory, a predecessor of the USAF Occupational and Environmental Health Laboratory (USAF OEHL), was tasked by the Air Force Surgeon General to develop an RF radiation protection program for implementation Air Force-wide.

During the period from 1965-1975 RF radiation protection efforts in the U.S. Air Force were governed by a medical directive, Air Force Manual (AFM) 161-7, Control of Hazards to Health from Microwave Radiation. The Permissible Exposure Limit (PEL) for occupational exposure to microwaves (RFR) was clearly established at 10 mW/cm^2 averaged over any 6 minute period. There was no specific provision for time-averaged exposures nor for consideration of scanning factors. The guidelines concerning the management of real or suspected overexposures to RF energy were ambiguous and contained no guidance or policies regarding overall management of the program or for making measurements when required.

Air Force Regulation (AFR) 100-6, Electromagnetic Interference and Radiation Hazards, contained then, as it does today, some delineation of responsibilities for field measurements. However, no provision existed for the nonmedical units with measurement capability/responsibility to effectively interface with the medical personnel who were not only responsible for base-level programs, but also for developing and setting PELs.

As a first order of business, personnel of the USAF Radiological Health Laboratory (USAFRHL) undertook to establish and solidify liaison with those nonmedical units who were doing field measurements. In late 1971 the principle organization so involved was abolished and the mission reestablished under the Electromagnetic Compatibility and Measurements (EMC&M) function of Headquarters Air Force Communications Service (AFCS). AFCS was later designated a Major Air Command and renamed the Air Force Communications Command (AFCC). This last action had no effect on the measurement mission, however.

About 1970, intense interest, Congressional and otherwise, was generated by allegations that radar operators had been exposed to hazardous levels of RFR which had caused cataracts while on the job in certain aircraft. The Air Force Medical Service found itself in need of measured data to support its medical evaluations to the contrary. As a result, in late 1970, under specific direction of the Air Force Chief of Staff through the Surgeon General, USAFRHL and AFCS conducted the first of a series of studies to determine if the allegations of RFR (microwave) cataractogenisis in fact had any basis. That first study lacked sophistication, but the investigators were unable to distinguish any difference in the eyes of career RF workers when compared to a matched group of non-RF workers.[1]

Shortly after that study was completed, the first of several revisions to AFR 100-6 was published, that more clearly defined the responsibilities for field measurements and how the data were to be disseminated to the medical community. It also, for the first time, assigned some field measurement responsibilities to the Air Force Medical Service. Also, at that time, work was begun to revise AFM 161-7 and reestablish it as a regulation with directive authority.

In 1972, interest was revived in microwave cataractogenisis by proposed Congressional Hearings on the matter. The Air Force medical community seized on that opportunity to

mount a more comprehensive study of the matter than was the 1970 effort. There had been two additional Air Force studies, similar in sophistication to the first, that were conducted with U.S. Army participation in 1971.[3,7] The 1972 study again included AFCS to provide measurements and involved nearly 1000 subjects. About one-half that number were in the study group and had worked in RFR occupations from 2 to more than 45 years. The other half formed a control group and were carefully selected for having had no occupational exposure to radiation and were just as carefully matched for age. The results of that so called "Five-Base Study" demonstrated that microwave cataractogenisis was a non-entity within the Air Force work force.[4,7] Primarily as the result of that study, microwave radiation was largely dismissed as a possible/probable cause of cataract activity among Air Force workers who are/were occupationally exposed at levels within the PEL.[7] That fact remains essentially true today.

In the fall of 1975, AFM 161-7 was rescinded and replaced by AFR 161-42, Radio Frequency Radiation Health Hazards Control, which provided for a more extensive RFR control program. Just before the appearance of the new regulation, decisions were made at the highest levels of the Air Force Medical Service that the program would be totally managed by Bioenvironmental Engineers at base-level. Consultant expertise would be developed at USAFRHL and efforts undertaken to acquire and maintain state-of-the-art instrumentation for loan to the field as needed.

AFR 161-42, also contained guidance and procedures for the management of overexposures to RFR. As a practical matter these procedures had been developed and loosely followed since early in 1972 when the first alleged overexposure was investigated and reconstructed. From that beginning, were developed the policies and procedures that were incorporated in AFR 161-42 in Nov 75.

As is often, if not usually, the case with fledgling efforts, the newly directed program was still somewhat hampered by inadequate authority to accomplish what needed to be done, by clear delineation of organizational responsibilities, and by deficiencies in awareness training of RFR workers. In addition, the Occupational Safety and Health Administration (OSHA), an arm of the U.S. Department of Labor, promulgated RFR Standards that were at slight variance with those of the Air Force. Those events led to still another revision of AFR 161-42 which was rescinded in 1978 when Air Force Occupational Safety and Health Standard (AFOSH Std) 161-9 appeared in October of 1978. That document served very well to guide the Air Force's program for several years but still had very basic inadequacies insofar as the management of overexposures was concerned. This was particularly true as regards where and by whom medical evaluations of individuals who were documented to have been overexposed would be accomplished. A current revision of AFOSH Std 161-9 was published in the fall of 1984 and essentially resolves virtually all of the program management difficulties that have been troublesome since 1970. It is the current guideline and authority by which the program is now managed and conducted. It also incorporates frequency dependent PELs from 10 KHz to 300 GHz. Under the authority of this Standard, personnel are trained and assigned a specific responsibility to maintain a high degree of safety in all RFR operations.

RFR Accident Investigations

Since the spring of 1972 the Air Force Medical Service has investigated RFR exposure incidents involving more than 330 individuals. Of that number 58 have been confirmed as overexposures and another 17 yielded inconclusive results, but were treated as overexposures as a matter of medical and legal prudence.

AFOSH Std 161-9 not only sets down the PELs for Air Force personnel, but it also specifically details how to prevent unnecessary or harmful exposures to personnel. In addition, it provides specific guidance as to what actions are required when an accident happens and/or when a real or suspected overexposure has occurred.

As previously noted, the RFR program in the Air Force is managed at base-level by the Base Bioenvironmental Engineer (BEE). These engineers have received at least some training in the management of RFR problems, and many have had extensive experience in the field.

Until 1979 very little RFR measuring equipment was available in the field and it was necessary for USAFRHL, and later USAF OEHL to either loan instrumentation to the bases which needed it, or to perform the surveys/evaluations themselves. That situation has markedly improved, so that today, over 40% of all Air Force bases have on hand adequate instrumentation for making field measurements of at least 95% of the emitters on any given base.

Since 1972, it has been Air Force policy that every suspected or alleged exposure to RFR in excess of the PEL be thoroughly investigated in order to: (1) positively determine whether or not an overexposure did occur; (2) if an overexposure did occur, to definitively determine both the power density level encountered and the length (time) of the exposure; and (3) recommend and coordinate appropriate medical evaluations should they be indicated.

The Standard requires that whenever a suspected overexposure occurs, or whenever an individual alleges one has occurred, the individual(s) involved must promptly report the matter to their supervisor. It is then incumbent upon the supervisor to insure that the individual(s) report promptly to an appropriate medical facility, usually the base hospital. The Standard also requires that Directors of Base Medical Services (DBMS), generally

the hospital commander, insure that their physician staff know and understand the principles of RFR injury and the appropriate tests and treatments that may be needed. If the RFR accident victims are in no obvious danger, the symptoms of any possible post-incident injury or illness requiring diagnostic evaluations will generally determine if hospital admission is necessary.

Air Force workers who are potentially exposed to RFR while on the job are also subject to the same illnesses and injuries that are typical of nearly all industrial workers. It is axiomatic then that lifesaving support measures appropriate to the presenting clinical symptoms must be given the highest priority. Most, if not all individuals overexposed to RFR at intensities less than those which are known to cause frank burns, will in all likelihood, manifest little or no immediate evidence of either physical distress or altered physiological function. The psychological/emotional reaction to the exposure, however, may be quite severe and may even require hospitalization for a short time for proper management and relief of the anxiety state commonly seen. The primary concern during the initial visit to the medical facility post-accident is to try and quantitate the exposure history in relation to any manifest symptoms and to document in detail certain medical baselines against which changes, should they occur, can later be measured. It is, therefore, imperative that a complete and comprehensive case record be established that will facilitate future decisions regarding the need for follow-up medical examinations and evaluation of the findings.

While the Standard specifically charges the DBMS with responsibility for initiating and conducting an investigation of all RFR accidents, as a practical matter, the Base BEE almost always acts in behalf of the commander in insuring that the proper actions are taken and that the documentation is complete. The investigating officer, who is also usually the Base BEE, must promptly gather the following background information concerning the accident/incident: (1) name, rank, and service number of all personnel involved; (2) RFR emitter nomenclature and operating parameters at the time of the incident; to include frequency, peak power, pulse width (PW), pulse repetition frequency rate (PRF), antenna characteristics, scan or rotation rate, beam configuration, etc.; (3) a description of what happened including date, time, place, duration and location and position of affected personnel in relation to the emitter in question. It is often possible to draw very significant but tentative conclusions at this point regarding the possibility/probability of an overexposure having occurred.

As soon as the salient facts surrounding the accident are known, the Standard requires that prompt notification of the matter be made to the next higher headquarters; e.g., Major Air Command (MAJCOM), usually the Command BEE. The Radiation Services Division of the USAF OEHL must also be notified. The USAF OEHL can and does provide expert guidance and assistance to the Base BEE in the conduct of the investigation, reconstruction and documentation of the incident. It becomes incumbent on the Base BEE at this point to decide whether or not he/she will do the investigation/reconstruction/documentation themselves or ask USAF OEHL to assume that responsibility. There is a relatively good case to be made for either approach, but generally it is felt that USAF OEHL is better able to assume the responsibility. There is, however, no requirement that it be done one way over the other.

The important point to be made is that it is absolutely imperative that the investigation be done just as soon as possible after the incident, primarily because the recall of those involved tends to become seriously flawed as time passes.

The incident must be meticulously reconstructed using the same emitter, operating at identical parameters, and at the direction of the personnel involved. The Standard is quite specific regarding these matters, even to including a discussion of radiation safety considerations for investigating personnel. Once the reconstruction has been completed, the data must be evaluated and a definitive determination made as to whether or not an overexposure did or did not occur. There are a great number of considerations that may come into play in making that determination, but those lie outside the scope of this paper.

If it is conclusively determined that <u>no</u> overexposure occurred, all medical activity connected with the incident is halted and detailed documentation to support that conclusion prepared. Copies are then distributed, including one permanently filed in the individual's medical record. Higher headquarters and safety offices are, of course, also included.

Occasionally, there are exposure incidents where the investigation is inconclusive as regards overexposure or no overexposure. This can occur where there are conflicting witness observations or perhaps when a really accurate reconstruction cannot be accomplished. There have been 14 such incidents in the Air Force since 1972 that involved 17 individuals. In such cases it is considered to be medically and legally prudent to treat them as though they were <u>over</u>exposures.

When it is conclusively determined that an overexposure <u>did</u> in fact occur or when the investigation is definitively inconclusive, there are a number of actions that are required by the Standard:

1. An accurate as possible quantification of the exposure
2. A determination as to what part of the body was primarily exposed, or was it a whole body exposure
3. A detailed review of any clinical symptoms manifested by the victims

4. Prompt consultation with Board Certified Occupational Medicine Physician(s) at USAF OEHL to determine what, if any, further medical evaluations are needed, and if so, where they will be obtained

The Air Force Standard also specifies in detail, what kinds of medical consultations/evaluations should be considered necessary under what kinds of overexposure situations. In addition, it outlines which medical offices/agencies are responsible for the professional and administrative management of these individuals. As a last point, all individuals who have been determined to have been overexposed to RFR, or those who are assumed to have been, are tracked throughout their Air Force career. They are periodically scheduled for medical reevaluation as appropriate to the magnitude of the exposure, etc. USAF OEHL has responsibility for this tracking procedure.

The U.S. Air Force RFR Accident Experience

In order to provide the reader with an actual frame of reference as to what kinds of alleged and confirmed RFR overexposures the Air Force has experienced, the following examples are presented:

Example 1: In June of 1974 while deployed in the field, a mobile communications technician was accidently exposed to RFR while attempting to connect a flexible wave guide to the output port of a TRC-97A tropospheric scatter unit.

The cause of this accident was never really determined, but the transmitter was operating in the CW mode at about 1 Kilowatt in the C-band (see Table 1). The technician's head was approximately 14 inches (35.5 cm) from the output port and he essentially looked directly into it. Within seconds the individual experienced intense subjective heating and within minutes was suffering from an acute anxiety reaction that required hospitalization and sedation. Within 30 hours, all symptoms had subsided and the individual returned to duty.

The reconstruction/investigation was conducted by USAFRHL and an exposure level of approximately 720 mW/cm^2 for about 30 seconds (21,600 mW-s/cm^2) was confirmed to have occurred. This exposure exceeded the existing PEL by a factor of 6. The victim was referred to the USAF School of Aerospace Medicine (USAFSAM) for complete medical evaluation. Over the next several years, this individual was reevaluated at USAFSAM on many occasions. Since the exposure was almost exclusively to the head, much attention has been directed toward the eyes. To this day the individual is well and the eyes unremarkable.

Table 1
Letter Designation of the Radar Bands[7]

Band	Frequency Range (MHz)
P	220 - 390
L	390 - 1550
S	1550 - 5200*
C	3900 - 6200*
J	6000 - 9000*
X	9000 - 10900
Ku	10900 - 22000
Ka	22000 - 36000
Q	36000 - 46000
V	46000 - 56000

*Note Overlaps

Example 2: In September of 1975 an autotrack (MSQ-46/M-33) radar technician was making precision calibration adjustments on the antenna, when the transmitter was inadvertently energized by another technician who was not aware of the calibration activities.

At the moment of exposure, the radar was operating in J-band at 350 KW peak power and a DF of 0.00025. The exposure was primarily to the scrotal area and the individual experienced only very mild subjective heating and was essentially unware of what had transpired until he had climbed down from the antenna and returned to the operations van.

The reconstruction/investigation was again conducted by USAFRHL at the request of the Base BEE. An exposure level of about 850 mW/cm^2 for 195 seconds (165,750 mW-s/cm^2) was confirmed to have occurred. The measurements in this case were very difficult to accomplish because of the extremely short DF of the autotrack. Because the exposure was more than 46 times greater than the existing PEL it is somewhat difficult to understand why the subjective reactions were so mild. The ambient weather conditions at the time of the incident, e.g., temperature 53°F (11.7°C), humidity 47%, barometer 30.01 inches Hg (750.25 mm Hg), wind 7 knots, overcast with light rain, probably were significant contributors. The individual was extensively evaluated at the USAFSAM with special emphasis on reproductive function. The findings were all unremarkable and the individual is apparently entirely well today.

Example 3: In March of 1978 three U.S. Army personnel were conducting a routine maintenance check on an MPQ-46 Hawk Illuminator, which was installed at an isolated U.S. Army site, but supported by a U.S. Air Force base nearby. No unusual circumstances were known to have precipitated the accident, other than simple carelessness on the part of the personnel involved.

The reconstruction/investigation was conducted by the USAFRHL at the request of the Base BEE charged with supporting the site. Since the Air Force had responsibility for site support, all elements of the Air Force RFR protection program applied. Of the three individuals involved, two were determined to have possibly been exposed to RFR levels of 90 mW/cm² for an indeterminate length of time, but certainly for longer than 6 minutes. Therefore, the existing PEL may have been exceeded. It was the opinion of the investigating officer that, in reality, the two were probably not overexposed at all because the evidence strongly suggested that the antenna was in motion during the entire time of alleged exposure. However, in the absence of reliable witnesses to that effect, it was elected to err on the side of conservatism and assume the worst case, e.g., the antenna was stopped and searchlighting the exposees.

The third individual was conclusively determined to have been exposed to 30 mW/cm² for 6 seconds (2100 mW-s/cm²), which was well below the existing PEL.

It was recommended to U.S. Army medical authorities that the two individuals who may have been exposed be evaluated by physicians who were familiar with RFR overexposures, etc.

Example 4: In October of 1978, an avionics technician was assisting with the checkout of an APQ-100 radar on an F-4C while parked on the flight line. The APQ-100 is a fire control radar operating in the low X-band with an average power of 110 watts. The technician was within approximately 1 foot (0.30 meters) of the antenna at the time of the incident.

The reconstruction/investigation was conducted by the Base BEE in consultation with the USAF OEHL. Because of the extremely high power densities encountered very close to such an emitter, it was impossible to accurately quantify the upper limit of the exposure, but a reasonable estimate based on experience and more distant measurements, was perhaps 400 mW/cm² for 240 seconds (96,000 mW-s/cm²). This exceeds the PEL by almost 27 times. The technician was acutely aware of a sensation of subjective heating. He also manifested a somewhat unusual skin rash over the upper trunk, head and neck for 2 to 3 days after the incident.

The technician was extensively evaluated at the USAFSAM and all observations were unremarkable. It was the USAFSAM physician's opinion that the skin rash was unrelated to the incident and it resolved without sequelae within 2 or 3 days. The technician is apparently entirely well today.

Example 5: In April of 1980 a USAF civilian avionics repair technician was conducting a final checkout of the avionics on an F-15A. He was not involved with the APG-63 radar, which was inadvertently energized while the technician was in front of the antenna. He promptly reported an intense sensation of heat to his head and neck and moved out of the beam.

The investigation/reconstruction was conducted by the USAF OEHL at the request of the Base BEE. There were a number of vagaries attendent to this incident and the investigation/reconstruction was quite complex. The ultimate determination, however, was that the technician was exposed to 550 mW/cm² for 15 seconds (8250 mW-s/cm²), which was more than twice the existing PEL.

The individual was extensively evaluated at the USAFSAM without significant findings and is apparently entirely well today.

Example 6: In September of 1983 eight civilian radar technicians were conducting antenna repairs and modifications on an FPS-92 tracking radar. Two of the technicians were U.S. Air Force civilians while the other six were employees of the USAF contractor responsible for the operation of the radar. The FPS-92, which operates in the mid-UHF region of the spectrum, was somehow energized while six of the eight individuals were working on the surface of the eighty-five foot (26 meters) diameter dish. At the time the average power output was between 100 and 150 Kilowatts.

The investigation/reconstruction was conducted by two RFR experienced BEEs who were stationed near the site, in close consultation with the USAF OEHL. These measurments revealed that two of the individuals were exposed to only a very small fraction of the PEL while the other six were exposed to power density levels ranging from 20 to 145 mW/cm² for eight minutes (480 seconds). These exposures are significantly in excess of the PEL.

All six of the exposees have undergone extensive medical evaluations at the USAFSAM and four have also been evaluated at one or more civilian institutions. The preliminary results obtained from the medical files at USAFSAM are inconclusive in that no findings were noted that could be directly attributed to the exposures, with the exception of acute situational anxiety reactions. All other manifestations were

viewed as being transitory in nature with no permanent effects expected. Reevaluations of these individuals are expected to continue on a regular basis for some years to come.

In each of the examples noted, plus all of the other incidents investigated, the results are meticuously documented and copies of that documentation made a permanent part of the individual's medical record. In addition, a more detailed record is also reposed at the USAF OEHL.

Summary of Medical Evaluation Results

Medical evaluations have been done on many, but not all of the personnel involved in RFR overexposures at Air Force bases since 1972. Not all of the personnel involved have been Air Force employees. There have been incidents on Air Force bases that involved civilian contractors, foreign nationals, U.S. Army and U.S. Marine Corps personnel.

In many cases the medical data obtained from the evaluations of the accidental RFR overexposures are incomplete in several respects, primarily due to a lack of standardization of the clinical examinations. Nevertheless, these case files can and do provide important anecdotal information concerning human exposure to RFR fields. This repository of case files is the only one of its kind known to exist.

Of the more than 330 (as of 1 Aug 84) suspected individual overexposure files in the repository, only 58 were positively confirmed to have exceeded the PEL. Of those 58, 26 individuals reported that they clearly felt a warming sensation at the time of the overexposure, 20 felt no warmth, and 12 were not sure. It can therefore be concluded that about 45% of those overexposed felt the energy and probably as a consequence of that feeling terminated the exposure. Of the approximately 240 alleged overexposures that were later positively confirmed as not exceeding the PEL, 26 felt a warming sensation and terminated the exposure before the PEL could be exceeded, 173 individuals felt no sensation and 39 were not sure.

Tables 2 through 7 summarize the accidental RFR exposures as a function of frequency, average power density, and exposure time.

Table 2
Confirmed Overexposures as a Function of Frequency

Number of Individuals	Frequency Range
1	20 MHz
7	200 - 500 MHz
18	1.5 - 6 GHz
24	8.0 - 10 GHz
5	15 - 35 GHz
3	Unknown

Table 3
Confirmed Overexposures as a Function of Average Power Density

Number of Individuals	Power Density Range (mW/cm²)
9	15 - 30
16	40 - 100
14	120 - 250
13	350 - 1,000
3	1,000 - 3,000
1	16,000 - 100,000
1	100,000 - 160,000
1	Unknown

Table 4
Confirmed Overexposures as a Function of Exposure Time

Number of Individuals	Exposure Time Range
7	1 - 10 secs
11	15 - 60 secs
18	1 - 6 mins
21	8 - 60 mins
1	Unknown

Table 5
Accidental RFR Exposures Within the PEL* as a Function of Frequency

Number of Incidents	Frequency Range
2	1 - 10 MHz
3	20 - 90 MHz
14	0.1 - 0.9 GHz
61	1.0 - 6.0 GHz
30	8 - 10 GHz
3	10 - 14 GHz
20	15 - 35 GHz
66	Unknown

*PEL = 3600 mW-s/cm² in any 6 min period.

Table 6
Accidental RFR Exposures Within the PEL*
as a Function of Average Power Density

Number of Incidents	Power Density Range (mW/cm²)
95	0 - 1
57	1 - 14
20	15 - 39
23	40 - 100
1	101 - 250
1	251 - 1,000
2	Unknown

*PEL = 3600 mW-s/cm² in any 6 min period.

Table 7
Accidental RFR Exposures Within the PEL* as a Function of Exposure Time

Number of Incidents	Exposure Time Range
29	0 - 1 sec
39	1 - 11 sec
36	15 - 60 sec
29	1 - 6 min
45	8 - 60 min
14	2 - 100 hrs
3	101 - 500 hrs
4	Unknown

*PEL = 3600 mW-s/cm² in any 6 min period.

Clinical Impressions

Medical review of the results of the physical examinations that were conducted following RFR overexposures have revealed few, if any, consistent clinical patterns. Even in the cases where very intense localized exposures occurred, erythema and/or edema were rarely seen at the time of the physical examination. Lenticular imperfections such as small punctate opacities and vacuoles were noted frequently in individuals whose overexposure was primarily to the head. However, none of these observations were felt to have been clinically significant since no concomitant impairment to visual function could be noted. Also, and very significantly, it has not been possible to reliably determine whether any of these imperfections were present in the individuals prior to the RFR incident. These same types of ocular imperfections are very prevalent in the population at large and often encountered during routine ophthalmological examinations.

Detailed psychological testing has been accomplished on a number of the overexposees. The evaluators have, on occasion, attempted to draw some conclusions, but those efforts are severely hampered, if not prevented, by the absence of pre-radiation baseline data for comparison and interpretation. It is important to note also, that no abnormalities were noted during the neurological examinations that were conducted in concert with the psychological studies.

In the entire overexposed group, serum enzyme levels, blood counts, blood pressures, sedimentation rates, and electrocardiograms were all judged to be unremarkable after clinical review by several physicians well experienced in the evaluation of RFR exposees. This, of course, is very strong suggestive evidence that no clearly defined tissue damage had occurred.

Individuals accidently exposed to levels of RFR at or above the PEL often manifest clinical symptoms that usually include headache, nausea, fatigue, malaise, palpitations,

etc. These symptoms can be attributed to an anxiety reaction to the exposure, but it is impossible to completely rule out an organic etiology. Some high level overexposures, e.g., >500 mW/cm², have resulted in anxiety reactions so severe that hospitalization and sedation were necessary. In some cases situational responses were severe enough to warrant psychiatric referral and evaluation.

Today, more sophisticated and formal review and analyses of these Air Force RFR accident medical files are underway. The general thrust of these analyses is toward evaluating the rate and type of clinical symptomology as a function of exposure level and frequency, and body part or area principally exposed.

Some Preliminary Conclusions

As the present ongoing review and analysis of the accident files continues, additional information will be forthcoming and perhaps some more meaningful and important conclusions can then be drawn. In the meantime, however, the following somewhat crude conclusions are evident at this time:

1. Of the nearly 330 alleged overexposures investigated, less than 20% were confirmed, the remaining ~ 80% were within the PEL.

2. About half of the overexposures were detected by the exposee because of a subjective heating sensation.

3. Virtually all of the overexposures were of a partial body nature.

4. Most of the exposures occurred at frequencies between 1 and 10 GHz.

The United States Air Force's experience with alleged overexposure to RFR has been extensive and has been well documented. Exhaustive investigations have been conducted and sophisticated medical evaluations of the confirmed exposee have been accomplished. To this point in time there is no hard or soft evidence to suggest that any permanent damage or injury has taken place in the individuals involved.

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Acknowledgements

The author wishes to thank Mr John Mitchell, USAFSAM/RZP, for his technical assistance, his patience, his understanding; and his staff for their statistical expertise and dilligence in extracting severely camouflaged data. A special thanks is extended to the Nonionizing Radiation Services Branch of the USAF OEHL for invaluable assistance in locating "lost" reports.

Review of Epidemiological Studies of Human Exposures to Radiofrequency Radiation

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Summary

The health effects of exposure to radiofrequency radiation (RFR) remain undefined and controversial. Epidemiological studies of human exposures to RFR are confounded by difficulties in determining the type and true extent of exposures, in selecting an appropriate control group for comparisons, in determining the existence and influence of many concomitant environmental factors, and in establishing the presence or measuring the frequency or severity of subjective complaints as well as objective findings in the studied populations. This paper reviews reported RFR effects on general health, growth and development, physiological systems such as the cardiovascular and nervous systems, and organs such as the eye. Criteria for reliable epidemiological studies are presented to allow critical analysis of such reports.

Introduction

The general public and, to a greater extent, occupational subgroups are being exposed increasingly to radiofrequency radiation (RFR). The health effects of such ubiquitous RFR exposure remain undefined and controversial. This is true despite the recognition of such exposures and concern regarding potential health effects, and despite the desires and efforts of many investigators and individuals charged with protecting or promoting the public's health and welfare. This paper will review the published data as well as interpretatations, by the investigators and others, derived from epidemiological studies as well as case reports of human exposures to RFR. Criteria for reliable epidemiological studies and clinical evaluation will be presented to allow discriminant evaluation of such reports.

Requirements for Valid Epidemiological Reports

The ability to ascertain, define and quantitate an effect is necessary for valid application of epidemiological methods to the study of an agent. There are numerous problems in designing epidemiological or incidence studies. It is especially difficult in regard to ubiquitous agents (particularly when exposures are hard to quantitate) to select paired populations that are not characterized by some other bias in regard to potentially interacting factors or exposures. The control or comparison group should be comparable to the case or exposed group in all relevant characteristics or circumstances except for the studied exposure itself. The sampled populations must also be large enough to make an increased risk of any effect detectable.

It is difficult to establish the presence as well as the frequency and severity of objective and especially subjective complaints attributed to RFR exposures. Eventually, if appropriate exposed and control groups are identified, if the exposures can be determined, if the bioeffects can be recognized and measured and statistically analyzed, and if the health implications can be ascertained, an epidemiological study may be developed that would be useful for setting exposure safety guidelines. To be thus useful, the report of the study should not only provide the data required to assess whether the study has met such criteria, but should also note any potential bias in subject identification, measurements, and other factors that may affect the inferences being derived regarding RFR effects.

General Comments on RFR Epidemiological Studies

An outstanding problem of epidemiological studies of RFR exposure is related to exposure assessment. Many published studies provide no documentation of the basic elements of exposures such as frequency, modulation, and power density. Often, populations are described merely as "exposed" or "non-exposed". Rigorous study designs, analyses, and discussions are often absent. Specifically, population selection criteria are usually not described and control groups are often not included or sufficiently identified. Control groups are occasionally described but inappropriate. Control of common confounding variables such as age or health status is commonly not considered. It is often necessary to study populations of many thousands in order to document or exclude significant results. The statistical methods applied are often not described, or may not be proper for the study design, failing to measure the strength of association via relative risk ratios. Many of the published studies are minimally descriptive or cross-sectional in nature. A majo limitation of epidemiological studies of RFR exposures is the lack of generally recognizable pathophysiological manifestations at realistic levels of exposure.

It is essential that multiple environmental factors, which can interact among themselves and with personal characteristics of the subjects, be evaluated. If reasonably controlled comparisons can be made for sufficiently long periods of time using groups that are comparable in important demographic, social and health characteristics, then possible health effects associated with exposure to RFR can be evaluated. In epidemiological studies, as in experimental or clinical investigation, there is rarely a single study, positive or negative, that can be accepted as definitive. Replication and validation are required, as well as recognition that inferences in regard to alternate conditions and exposures are unsubstantiated, although usually necessary because of the limits (cost or otherwise) upon investigation.

Review of Representative RFR Epidemiological Studies

Among the effects attributed to RFR exposure, by published case reports and occasionally by epidemiological investigations, have been the development of cataracts, cancer including leukemia, male

infertility, congenital defects, and various functional disturbances. Not unexpectedly, reports claiming a link between RFR exposure and adverse effects on health have generated more publicity and received more widespread attention, mainly in the popular press but unfortunately also in uncritical scientific reviews. For example, an early widely known case-control study claimed an association between prior exposure of fathers to RFR and mongolism in offspring (1). In contrast, a subsequent less well known report by the same investigators extended the evaluations to a greater number of subjects, with better accumulation of information regarding RFR exposure, and concluded that there were no demonstrated statistically significant differences in risk of mongolism in offspring between RFR-exposed and non-exposed fathers (2).

Numerous studies of occupationally exposed individuals suffer from serious limitations. Exposure conditions are not defined, due to necessarily inadequate knowledge of essential parameters such as power densities and durations of exposure, effective area of irradiation, fluctuations in pertinent environmental factors, etc., as well as individual variations in threshold for or magnitude of induced alterations in health and well being. Commonly, the only parameter available (although not always provided) is the range of power densities of RFR which might have been encountered over the period of investigation.

General Effects on Health. A large number of surveys of occupationally exposed workers have been conducted in the Soviet Union and other Eastern European countries, and a relatively limited number conducted in the West. Rarely, East and West are involved, although with variable consent, in determining potential effects of exposing individuals to RFR, notably the widely publicized Soviet microwave beam directed at the US chancery building in Moscow. Exposed rooms, directly in the path of the beam, were found to have a higher power density of RFR enengy than is usually found at ground level near transmission towers. The health of foreign service and other employees and dependents who had worked in Moscow and other selected East European facilities over more than two decades were studied to ascertain any adverse effects on the Moscow workers presumably due to RFR exposures (3,4). A sufficient number of subjects had been in the exposure population in the distant past (two decades previously) to allow potential delayed effects to develop. Analyses of the exposed and non-exposed populations revealed no differences in health status as indicated by mortality rates or a variety of measurements of morbidity. The report stated that "No convincing evidence was discovered that would directly implicate the exposure to microwave radiation experienced by the employees at the Moscow embassy in the causation of any adverse health effects at the time of this analysis." Despite the careful review of the epidemiological data, from an event or situation that more closely allowed quantitation of exposures than is commonly the case, the exposures of the Moscow embassy employees continue to be linked in the non-scientific literature with sporadic adverse effects on health. Such connections have been effectively dismissed by scientific nalysis (5,6).

Another extensive study compared 20,000 military electronics technicians, including individuals who may have had substantial exposures to RFR, with an equivalent number of personnel assigned to other duties, and noted no differences in long-term mortality or rate of hospitalization around the time of the exposure (7).

Effects on Growth and Development. The potential effects of RFR exposure on human growth and development remain controversial, and assessment of the varied reports demands considerable circumspection (8). For example, RFR diathermy has been used by some for treatment of various pelvic conditions even durng pregnancy, and while some women thus treated have had offspring with congenital defects (9,10), others have had no difficulties with conception, pregnancy, or well-being of offspring (8,10). In one case, for example, a woman aborted in the first trimester while being treated with RFR diathermy, but delivered a normal baby after a subsequent pregnancy during which she again underwent RFR treatment (10).

While a report concluded, after a study of approximately 30 workmen, that long-term exposure to RFR could induce alterations in spermatogenesis (11), other investigators found no changes in fertility of RFR workers (12,13). As noted above, an initial impression that RFR exposures of fathers could be associated with mongolism in offspring was not confiremd by further, more extensive and careful investigations (1,2).

One report suggested an excessive incidence of congenital abnormalities occurring in communities surrounding a military base, with a large number of RFR (radar) sources in the vicinity (14). The conclusion that the rate was excessive was based upon comparison with rates derived from state birth certificate notifications. A more detailed investigation suggested that there was excessive reporting from the military base itself, with errors in the malformation data. It was concluded that there was no convincing evidence that radar exposure was related to congenital malformation (15).

RFR heating has been used to relieve the pain of uterine contractions during labor (16,17). In 2000 selected patients without obstetric pathology, the babies were born healthy, with good circulation and without evidence of physical injury or mental retardation over a year of follow-up. However, it must be noted that gross structural defects would be unlikely to develop with exposure of a human fetus at this late stage, since it is almost fully developed at parturition.

Effects on Cancer Incidence. RFR-induced cancer has not been demonstrated in medical surveillance examinations of RFR workers or military service personnel (18,19). Although the popular press occasionally asserts a link between exposure to RFR and the development of cancer, two cohort epidemiological investigations (3,7,20) that studied the question systematically did not show an excess of any form of cancer that could be connected to RFR exposure (18,19). Furthermore, with ten years or more of follow-up of large groups of subjects occupationally exposed to RFR of varying power densities, including relatively higher density exposures, there were no reports of malignancies of any type (21,22).

Cardiovascular Effects. A set of labile, possible functional cardiovascular changes (sometimes in opposing directions) have intermittently been atrributed to RFR exposure. Potential effects indicated by hypotension, bradycardia, delayed atrial or ventricular conduction, and electrocardiogram alterations among workers in RFR fields have been reported (23-26). Such changes have not diminished the capacity

to work, and have been reversible (27), and no identifiably serious disturbances have been noted as a result of RFR exposure (28).

Effects on the Nervous System. Clinical and laboratory studies of workers in the Soviet Union and other East European countries employed in the manufacturing, testing, maintenance, and operation of RFR-generating equipment have described central nervous system effects of RFR exposure (21,24,26,29-33). According to some of the reports, exposures have been mainly low-level (less than a few milliwatts per sq. cm.) and long-term. With few exceptions (34), functional disturbances have been described as a characteristic RFR syndrome termed the "neurasthenic" or "asthenic" syndrome. The symptoms and signs included in the syndrome are generally rather subjective, such as headache, fatigability, irritability, loss of appetite, sleepiness, sweating, difficulties in concentration or memory, and depression or emotional instability. The syndrome is said to be reversible in most cases if exposure is discontinued (18,19,26, 35). Furthermore, there do not appear to be significant pathological changes in neural structures (35). Several reviewers (8,18,19,21,36-40) have emphasized the difficulties in establishing the presence of or quantifying the frequency or severity of such relatively subjective complaints.

Ocular Effects. There has been great interest in alleged cataractogenic effects of exposure to RFR. Numerous surveys of potential ocular effects of RFR exposure have been made, commonly involving military personnel or civilian workers at military bases or in industrial settings (18,19). Several cases of cataracts have been attributed to RFR exposure (41-43), but such an association has not been confirmed (44,45).

No distinct morphological features distinguish lens opacities in RFR workers from those seen in a control population (46-48). While alleged "RFR cataracts" do not appear to be distinguishable from other cataracts (49-52), one author (Zaret) asserts otherwise (43). That author's claim that there are lens changes recognizable as RFR-induced has been disputed by many (45).

Most surveys, in contrast to case reports, have examined minor lens changes (not affecting visual acuity) in exposed and control groups (44), even though there is no evidence from ophthalmological reviews that minor lens opacities are precursors of clinical cataracts (18,19). A case-control epidemiological study of war veterans did not show an association with the development of cataracts (53). A few suggestive differences have been reported (54-56), but there is no clear indication that minor lens defects are a marker for RFR exposure in terms of types or frequency of changes, exposure factors or occupation (18,19).

As with reports of other RFR effects, it is difficult to adequately assess the reported earlier appearance of lens defects in RFR workers than in comparison groups because of considerable variation in the type, number, and size of defects recorded, in the scoring methods used by different observers, and in the numbers of individuals examined (8,18,19,44). Neither definitions nor methods of detection and measurement of cataracts are standardized (18,19).

Concluding Remarks and Recommendations

The available data regarding human exposures to RFR are almost exclusively derived from uncontrolled observations. Although controlled exposures have been used for therapeutic purposes, the conditions (commonly intense exposure of a limited part of the body for short durations) are not representative of general public or specific occupational exposures. Nonetheless, such therapeutic exposures can provide some indication of tolerable exposure levels under specific and well-defined conditions.

In virtually all of the published epidemiological studies and case reports, exposure conditions have been largely undefined or loosely inferred. The most severe limitations upon interpretation of these reports must arise from the fact that actual power levels and durations of RFR exposure are unsubstantiated. There has usually been inadequate consideration of multiple environmental factors which can interact among themselves as well as with the biological or behavioral characteristics of the individual subjects.

Further careful analysis of potential RFR effects are most likely to be forthcoming from studies with occupational exposures to RFR, although evensuch analyses are encoumbered by many difficulties (8,57). For example, the "adequacy" of control groups is often arguable, and the quantitation of occupational exposure is extremely difficult. The latter is particularly true when the subjects are mobile in the course of their exposures, and are exposed to non-stationary or varied types of fields.

It must be kept in mind that alterations in function or in bodily health are commonly not due merely to the presence or absence of a single factor. Such events are arrested, modified, or enhanced by multiple personal (or constitutional) and environmental characteristics, which may influence each other as well. Thus, detectable RFR effects, where substantiated, may have different health implications depending upon whether physiologically possible or impossible adjustments are required for homeostasis. Depending upon the circumstances, what may be an adverse effect for one individual may be beneficial to another.

Because of the difficulties in extrapolating from animal experiments to human effects, and despite the difficulties noted above, epidemiological studies, with appropriate correlative or mechanistic clinical and laboratory examinations, are essential to further our understanding of possible health hazards associated with exposure to RFR. The development and application of instrumentation for more accurate and complete measurement of exposures merits particular attention. Field strength, electrophysical, and thermal probes which provide artifact-free recordings without distortions of the field or inadvertant stimulation of tissue (due to induced currents) are essential.

A careful search should be made for available exposed (and control) groups not yet examined. Studies of occupational or other exposed groups, as well as control groups, should include a thorough analysis of the exposure (and sham-exposure) environment, with personal as well as common potential cofactors considered in determinations of RFR effects. The presence of multiple possibly confounding factors probably

can not be escaped, and is in fact important for application of any experimental or investigational observations to the setting of real life.

It is important to maintain a proper scientific perspective and assess realistically the biomedical effects of RFR exposure, so that the occupational or other groups specifically under consideration, and the general public will not be unduly exposed to a hazard, nor denied access to the benefits that may be derived from appropriate research, development, and utilization of RFR energies.

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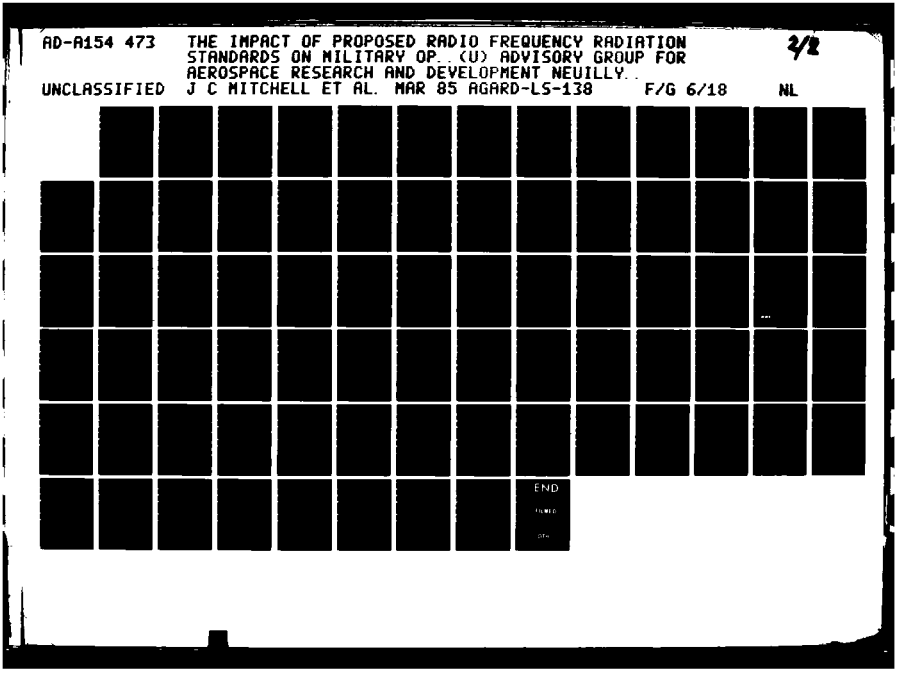
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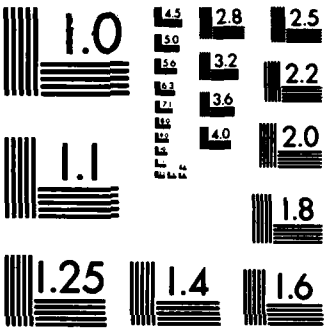

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Acknowledgements

The authors' work is supported by contracts or grants from the US Air Force School of Aerospace Medicine (F33615-84-C-0608), the US Department of Energy (DE-AC02-76EV03490), the National Institute of Allergy and Infectious Diseases (AI 15547) and the National Institute of Environmental Health Sciences (ES 03239).

EVALUATION OF HUMAN EXPOSURES TO LOW FREQUENCY FIELDS

by

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SUMMARY

The biophysical model concept described in this paper might be suited as a basis of discussion to determine and define limits of exposure to electric or magnetic fields below 100 kHz, including 50/60 Hz.

The electric field strength within the tissue in the environment of excitable neurons and muscle cells is considered decisive for the biological effect. Threshold values of field strength or current density, inducing biological effects are compiled from experimental and theoretical studies. On the basis of these data it is possible to establish "safe", "dangerous" and "hazardous" current density curves as a function of frequency. The criterion for the definition of injury is the elicitation of ventricular fibrillation which must be avoided. To define exposure limits, the field strength or current density causing injury should be reduced by a factor exceeding 100. The arguments supporting this wide safety margin are discussed.

In the second part of this paper the electric and magnetic field strength in the human environment is correlated with the corresponding electric current density induced in the human body. This enable "safe", "dangerous" and "hazardous" levels of current density in the human body to be correlated with the external electric or magnetic field strength. Parts of the concept presented in this lecture have been adopted as the scientific basis for the lower frequency range of the standard DIN 57848/VDE 0848: Hazards by electromagnetic fields; Protection of persons in the frequency range from 10 kHz to 3.000 GHz.

1.0 INTRODUCTION

In the course of the past few years the experimental and theoretical bases were reviewed which allow assessment of a potential hazard to personal health by electromagnetic fields (IRPA/INIRC, 1984; NCRP Report 67, 1981; Suess (ed.), 1981; UNEP/WHO/IRPA, 1981; UNEP/WHO/IRPA, 1984). Limits of electric and magnetic field strengths are taken into account or submitted as drafts in many countries for protection of personnel or individuals from the general public. What is conspicious is the fact that, in the majority of the regulations, the applicable frequency range does not start below 0.1 or even 1 MHz. There is only a very small supply of experimental and theoretical data on the specific field strengths that can be tolerated without detriment to health for the frequency spectrum between 1 Hz and 100 kHz (with the exception of the fields common in power engineering). Persons may be exposed, however, to strong fields with frequencies below 100 kHz in fields other than the application of power engineering (16 2/3, 50, 60 Hz), with magnetic fields being of specific interest on account of their penetration characteristics for the human body. Various types of induction heating systems in the low- and medium-frequency range are examples of sources of strong magnetic fields. Biologic effects occur with a sufficient intensity of the fields. The examples include the hair movement on the body in strong electric fields or the generation of light and flickering phenomena and subjective complaints such as headache in strong magnetic fields. In medicine, the effects of strong magnetic fields are being made use of in imaging processes (nuclear spin tomography) or for therapeutic purposes (magnetic field treatment). Biological effects and health risks from fields occuring during NMR application were compiled by Budinger (1979, 1981), Saunders (1982) and Bernhardt & Kossel (1984a, b).

The reason for the uncertainty in determination of personnel health limits for frequencies below 100 kHz is the fact that so far a sufficiently secured model idea has not become known in the estimation of the risk in the frequency range involved here. Such a concept, however, is necessary as one cannot expect that the full frequency range can be studied by experiments with good results similar to those obtained in the sphere of power engineering fields. This paper describes a simple concept which may serve as a basis in the discussion on the definition of personnel health limits. Parts of these ideas and considerations have already been adopted in the VDE regulation (1984) defining limits for frequencies above 10 kHz. The same considerations are also employed in the "Safety regulations for working places with risks of health hazards by electromagnetic fields", issued by the trade association in which limits of the electric and magnetic field strengths above 1 kHz are set forth (Specification for accident prevention, 1982). The concept described here continues the considerations published formerly (Bernhardt, 1979, 1983a, b, 1984b).

2.0 BASIS OF THE CONCEPT FOR EVALUATING HUMAN EXPOSURES TO LOW FREQUENCIY FIELDS

Differently to the static magnetic fields there are no indications for time varying magnetic fields that there exist a direct specific magnetic field effect at tissue field strengths below that value at which induced eddy currents may cause biological effects (Faraday's law).

Therefore, when possible health risks from the influence of electric and magnetic fields on man are evaluated primarily those biologic effects are considered which originate from a direct action on the cells in nerve and muscle tissues. The physical quantity determining the biological effect is the electric field strength in the tissue surrounding the living cell. This can be inferred from both theoretical considerations where the depolarization of the cell membrane potential is directly related to the magnitude of the electric field strength in the cell environment, and from the experiments confirming this concept. A great volume of experimental data on stimulus thresholds for different nerves and muscle cells, however, has often been expressed in the form of electric current values and not as field strength values. There are only very few papers which disclose data on the field strength thresholds, more data, however, exist on current density thresholds. Therefore, the electric current density will be employed as the decisive

parameter in assessment of the biologic effects at cell level. As far as necessary, the values given for the specific conductivity can be employed to convert the current density in the tissues into field strength.

Selection of the current density as a measure of an action on the cellular level also offers the possibility to extrapolate conditions in the human body from studies of animal experiments or from measurements taken at isolated cells, by way of mutual comparison of the current densities. It is important that it is irrelevant whether the electric current density surrounding a cell is introduced into the body through electrodes or induced in the body by external electric or magnetic fields, however, the current paths within the body may be different.

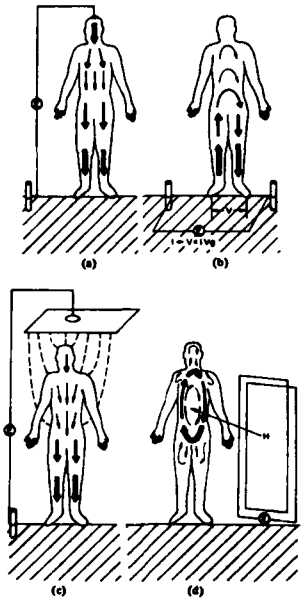

Fig. 1: Internal current density distribution for an erect primate. (a)-(d) indicate four possible ways wherein current can be caused to flow in an erect primate. The direction of arrows indicates the approximate direction of the internal body current flow and the width of the arrow suggests the current densities. The paths for displacement current flow are suggested by the dashed lines. (a) Conduction case. (b) Step potential case. (c) Electric field induced flow. (d) Magnetic field induced flow (from Bridges & Preache, 1981).

This is illustrated by Fig. 1 which indicates four possible ways in which currents can be induced to flow in an erect primate (Bridge & Preache, 1981). Various current paths are possible therefore "worst case"-considerations are necessary. The figure illustrates that current values which are used to evaluate the effects of body currents in the conduction case, are not the suitable quantities to evaluate the exposures to electric or magnetic fields. The evaluation of field exposure is additionally made more difficult because the exact current paths depend on the conductive properties of the body tissues in a complicated way. Therefore, often only rough estimations are possible.

In the evaluation of human exposure to electric and magnetic fields below 100 kHz, the following steps are relevant:

a) The experimental data on the thresholds for stimulation of excitable cells are combined in a current density/frequency diagram. A current density "envelope" is employed as the "threshold curve of possible acute health hazard" while another current density curve is plotted as the "injury threshold".

b) Some experimental values in relation to phenomena depending on current densities below the stimulus thresholds, in combination with theoretical considerations, define a current density curve below which a direct influence on neurons can no longer be expected ("limit of the safe range").

c) There is the current densitiy curve, between the "safe" and the "dangerous" current density curves, which may serve as the limit value curve in evaluation of the exposure to external electric and magnetic fields.

d) Electric and magnetic field strengths in man's environment are related to the electric current densities they induce within the human body. This allows correlation of the internal current density curves with the external field strengths and to define "safe" and "dangerous" field strengths.

e) It must be verified that there exist no other direct or indirect biological effects caused by other mechanisms which could lead also to a hazard of man at lower field strengths than those defined in d).

3.0 EXPERIMENTAL DATA FOR DEFINITION OF "SAFE" AND "DANGEROUS" CURRENT DENSITIES

In this chapter threshold values of the electric current density for different biological effects in nerve and muscle tissue are considered. The values are summarized in current density/frequency diagrams (Figures 2 and 5). Some publications have been selected from numerous studies on the fields of biophysics, electro-physiology, medicine and electrical sciences (electrical accident), from which threshold values could be derived for the electric current densities for stimulation of neurons or muscle cells as well as frequency data.

3.1 Stimulation Thresholds

3.1.1 Stimulation of Sensory Receptors

There exist reliable quantitative data about human thresholds of electric shock at power transmission frequencies. Three physiological responses to electrical stimulation had been investigated: perception, uncontrollable muscular contraction, and death (Keesey & Letcher, 1970; Dalziel, 1972). The thresholds for stimulation of nerve, skeletal muscle, and cardiac muscle were found to be minimal in the frequency range between 10 and 100Hz. Looking for perception thresholds, the sensation thresholds depend upon the location selected on the body and the nature of the contacts made. Some individuals, for instance, can detect currents of 5 - 10μA with the tongue. This is corresponding to current density values of 10 to 100μA/cm². When the perception threshold was determined by a finger-tapping contact on a flat electrode, the touch perception for 50 % of women was 0.24 mA, that for 50 % of men 0,36 mA(Dalziel, 1954a, b). In IEC Publication 479 (1983) the perception threshold was set to 0.5 mA. Referring to current density values, an investigation of Geddes et al. (1969) may be used. They applied various electrode sizes and arrangements on test subjects to measure the sensory receptor stimulation over a wide frequency range. Information on current densities can be given because the stimulation takes place immediately underneath the electrodes. Curve (a_1) in Fig. 2 interlinks values measured with an electrode arrangement applied to the test subject throat and venter, whereas for curve (a_2) a transthoraic electrode arrangement was used. Values measured by Dalziel (1954a) show a similar frequency response, but they do not allow any numerical data on current density. Approximately, above 100 kHz the tingling or itching sensation turns into a sensation of heat.

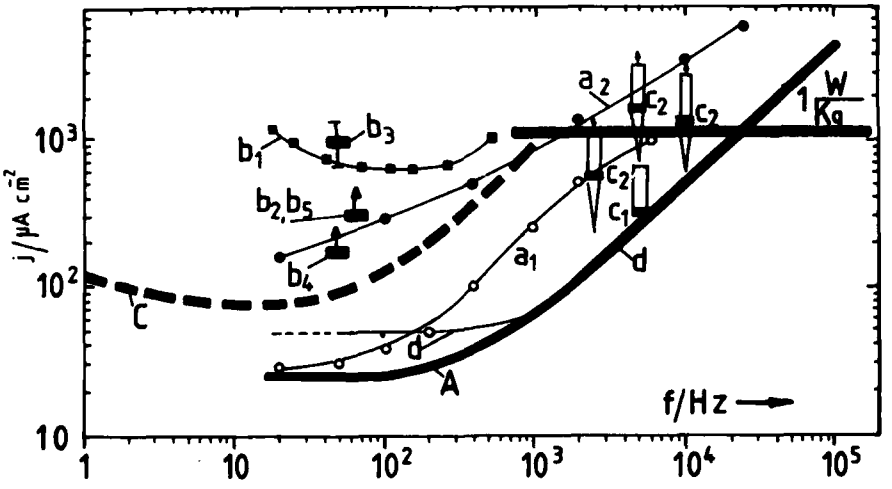

Fig. 2: Threshold values of the electric current density for different biological effects.
- a_1, a_2: Stimulation of sensory receptors immediately underneath surface electrodes;
- b_1 - b_6: ventricular fibrillation thresholds for stimulation times of 1 second or longer (different authors, see text);
- C: threshold curve for extra-systole elicitation, stimulation 1 second or longer;
- c_1, c_2: extra-cellular stimulation thresholds of single cells,
- d: frequency dependence of stimulation thresholds for nerve/muscle systems;
- A: "envelope" threshold value curve for stimulating effects.

3.1.2 Disturbance of Cardiac Stimulation

When the electric fields intensities in the environment of myocardial cells are sufficiently high the process of intracordial stimulation can be influenced. Two processes are relevant here: the occurrence of extrasystoles and the triggering of atrial and ventricular fibrillation of the heart. Whilst premature heart contractions in the course of the regular pulse sequence are deemed disturbances of the cardiac stimulation the ventricular fibrillation is the most frequent acute cause of death in the electrical accident. During fibrillation, a chaotic activity takes place of the well-ordered process of cardiac stimulation, with the result that the ventricles can no longer be filled and emptied. The consequence is a drop in blood pressure with arrest of the blood circulation.

Even though there are numerous studies on current intensity, duration of exposition and current path in electrical accidents (e.g. Brinkmann & Schaefer, 1982, and literature quoted ibidem), information on the field strength or current density values leading to disturbance in cardiac stimulation can hardly be found.

In view of the "worst case" event it is important to know that the risk of ventricular fibrillation increases with the duration of the current flow. Measurements made by Roy et al. (1977) on dog hearts with catheterized intracardial stimulation of 60 Hz have shown that with a current flow duration of a single period more than 100 mA are necessary to elicit fibrillation whereas with a duration of 100 periods the fibrillation threshold ranges about 0.7 mA (Fig. 3). Therefore, the further statements will be based on the exposition times of 1 second or longer.

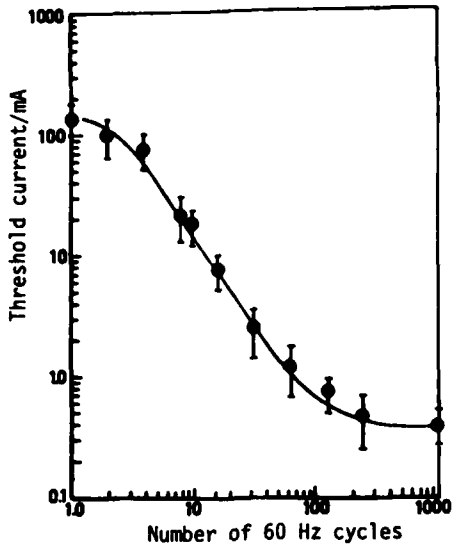

Fig. 3: Average values of current fibrillation thresholds (from Roy et al. 1977)

Thresholds for fibrillation elicitation are higher than the stimulation thresholds for extra-systole elicitation, by the factor of 3 to 5 (Antoni, 1982, Weirich et al., 1982), depending on the respective frequency. When extrasystoles occur at ever increasing frequency as long as stimulation lasts, a change-over to ventricular fibrillation eventually will be noted. The studies on the frequency dependence of the diastolic stimulation threshold and the ventricular fibrillation threshold with sinusoidal AC in the frequency range from 1 to 1000 Hz, as carried out by Antoni et al., are deemed fundamental in assessment of disturbance in cardiac stimulation. It is important for evaluating that the biophysical basic mechanisms involved in creation and further course of ventricular fibrillation in the studied animal hearts are the same as the mechanisms to be found in the hearts of bigger animals. So information on the electric field strengths or the current densities in the tissue allow the interpretation of results obtained in animal experiments to the human body. The range from 100 to 1000$\mu A/cm^2$ is considered to be the threshold of current densities in the tissues which elicit ventricular fibrillation at 50 or 60 Hz (Schwan, 1977). While attempting to define this range somewhat more exactly, the following thresholds of current densities for ventricular fibrillation were found:

Irnich et al. (1974) measured the stimulation and fibrillation thresholds with dogs and recorded these values as a function of the frequency (16 - 300 Hz). It is possible to read current density values from the information given in that publication on the field strength eliciting fibrillation with a continuously applied sinusoidal voltage (assumed conductivity 0.25 S/m, curve b_1 in Figure 2).

The threshold values found by Roy et al. (1976) with cardiac catheters are above 300 $\mu A/cm^2$ (b_2 in Figure 2) - depending on the catheter size.

Jacobsen et al. (1974) measured the field strength at pig hearts, quoting a confidence range for the electric field strength threshold to elicit ventricular fibrillation between 224 and 429 mV/cm (mean value 327 mV/cm). The current density values converted for a myocardial conductivity of 0.25 S/m are plotted under b_3 in Figure 2.

Osypka (1963) quotes a threshold value of 8 V/m which is sufficient to start the stimulation process at the heart. Calculation and conversion lead to a current density of 2.0 A/m^2 (200 $\mu A/cm^2$, b_4 in Figure 2).

Studies carried out by Watson et al. (1973) with the human heart, using 1.8 cm^2 electrodes, result in a threshold of 300 $\mu A/cm^2$ to elicit ventricular fibrillation (b_5 in Figure 2).

Kugelberg (1976) demonstrated the frequency dependence of the fibrillation threshold with 3 patients during an operation at the open heart (frequency range 8-800 Hz), with the threshold current intensity minimum, that elicited ventricular fibrillation, ranging between 20 and 60 Hz; at 800 Hz current intensities were required that were roughly eight times higher.

The current values quoted by Weirich et al. (1982) for the stimulation threshold for extra-systole elicitation plotted in Figure 2 as current density curve in a manner that the curve (C in Figure 2) is running by a factor 3 to 5 below the fibrillation thresholds. Curve C in Fig. 2 is corresponding to the time-current curve C_1 in Fig. 7 for current flow times of 1 s or longer.

3.1.3 Stimulation of Isolated Cells

Using micro-electrodes, several authors measured the thresholds of the current for extra-cellular stimulation of isolated neurons, with varying spacing of the micro-electrode from the cells. Current density values for the stimulation thresholds can be calculated from these current intensity/spacing measurements. The stimulation experiments were carried out with isolated rectangular pulses of short duration

(0.05 to 0.2 ms); for reasons of simplicity, their effects were equalled to the effect of isolated sinus alternations. Simulation is governed by the spacing between the stimulating electrode and the next node of Ranvier. Unfortunately, this spacing is indicated in one paper only (Roberts & Smith, 1973), there is obtained a minimum threshold current density of 0.28 mA/cm² with 0.1 ms duration of stimulation (c_1 in Fig. 2). Other authors indicate only the shortest microelectrode-cell spacing, which was then taken as a basis for the calculation of current densities. In these calculations it was assumed that the current would spherically propagate from the tip of the microelectrode.

There is a great variability between the individual studies, which is possibly due to different spacings from the next node of Ranvier. Some of these measurements have been interpreted (Ranck, 1975, c_2 in Figure 2), however, considering only those test points which are located at a wide distance between the electrode and the cell. Therefore, the spacings from the next node of Ranvier are at least of equal distance or even longer, i.e., the current densities determined are equal or even smaller than those quoted (indicated in Figure 2 by the reduction of the bars towards the smaller current density values).

3.1.4 AC Stimulation Threshold

Schaefer (1940) quoted a formula to express the stimulation threshold for nerve/muscle systems as a function of frequency. The formula gives the stimulation threshold of alternating current in the form of a threshold ratio to the 50 Hz threshold for different frequencies. There is a linear threshold increase with a frequency increase in the high frequency range. Here reference is made to current density values. When a 50 Hz threshold of 0.5 A/m² is selected, the current density ranges for isolated-cell stimulation, designated by (c_1, c_2) in Figure 2, are just above that curve (curve d in Fig. 2). Dalziel (1954b), too, concluded from his measurements of the perception thresholds that the threshold currents at 10 kHz are about 10 times the threshold currents at 60 Hz.

3.1.5 Induction of Membrane Potentials by Electric Fields Surrounding a Cell

This Chapter is given to demonstrate that a threshold value for stimulating effects of roughly 300 µA/cm² must also be expected for theoretical reasons.

As a result of charge transfers inside and outside the cells, alternating electric fields induce a membrane potential in biologic cells, which is determined not only by the field strength and the frequency of the field, but also by the size and shape of the cell as well as the cell orientation in relation to the electric field (Bernhardt, 1973).

The field-induced membrane potential reaches a maximum in elongate cells extending in parallel to the direction of the field. The maximum value V_m is reached in this case at the ends of the cell (length L) and can be defined by the following relationship (cf. also insertion in Figure 4):

$$V_m = \frac{1}{2} \cdot L \cdot E \cdot \left(\frac{1}{\sqrt{1+\left(\frac{f}{f_o}\right)^2}} \right) \qquad (1)$$

wherein:

E = electric field strength external of the cell

f_o = frequency for which applies

$$V_m = \frac{1}{2\sqrt{2}} \cdot L \cdot E = V_m^0 / \sqrt{2} \qquad (2)$$

The correlation between f_o and the cell type has not yet been studied by way of experiments. With sufficiently low frequencies $f \ll f_o$ applies:

$$V_m^o = \frac{1}{2} \cdot L \cdot E. \qquad (3)$$

This theoretical value could be confirmed by measuring the field-induced potential difference at plant cells, using intracellular micro-electrode tracing. In these measurements, plant cells of different lengths (Nitellopsis obtusa) were placed into artificial pond water and exposed there to electric fields of varying strength and frequency. The internodal cells used had a length between 1.5 and 11 cm, their diameter varied from 0.2 to 0.8 mm. The cells were fixed in a long Plexiglas chamber whose ends were provided with two electrode plates to produce the electric field in parallel to the longitudinal direction of the cell. The potential distribution inside the chamber was measured with a sliding-type micro-electrode filled with an electrolyte. After the cell had been pierced with the micro-electrode the potential difference V_m could be measured at the ends of the cells as a function of the field strength and the frequency.

The values measured confirmed the theoretical value V_m^o that had been found for short cell lengths up to 2 cm: with an increasing cell length the lag behind the theoretical value increased, too (Fig. 4). For the presentation of the results in Fig. 4, E is chosen in such a way that L · E = 100 mV.

It is of particular importance, however, that measurements could be taken with these cells in solutions having different electrical conductivity characteristics. It turned out that the field-induced potential differences at the cell membrane are a function of the field strength in the external solution only, and not of the current density in the environment. This is the experimental proof of the induction of potential differences being a field strength effect. In relation to the considerations made in this paper, this means, that in cases where current density effects, for instance, are studied with different specific

conductivity characteristics a conversion is required into the relevant electric field strength, via the conductivity. From eq. (3) the field strength in the cell environment can be calculated for a variation of the membrane potential by 10 mV, and the corresponding electric current density can be calculated from the relationship $j = \sigma \cdot E$. When a Ranvier node spacing of roughly 2 mm is assumed for the effective cell length (BeMent & Ranck, 1969), a current density of 2.5 A/m² (0.25 mA/cm²) arises for a variation of the membrane potential by 10 mV at the cell ends when $\sigma = 0.25$ S/m. For instance, a depolarization by 10 to 20 mV, approximately, may result in a stimulation effect in excitable cells if during such depolarization the threshold charge quantity which is necessary for stimulation is transported through the cell membrane.

It must be assumed, however, that for ganglion cells other threshold values for the current density are necessary, i.e., probably higher than those for the fibrelike cells, like nerve and muscle cells as considered here. Generally, one has to start from the principle that the stimulation thresholds for small cells may be considerably higher. The simple model described in this chapter can only give the range of order for threshold values with regard to the "worst case".

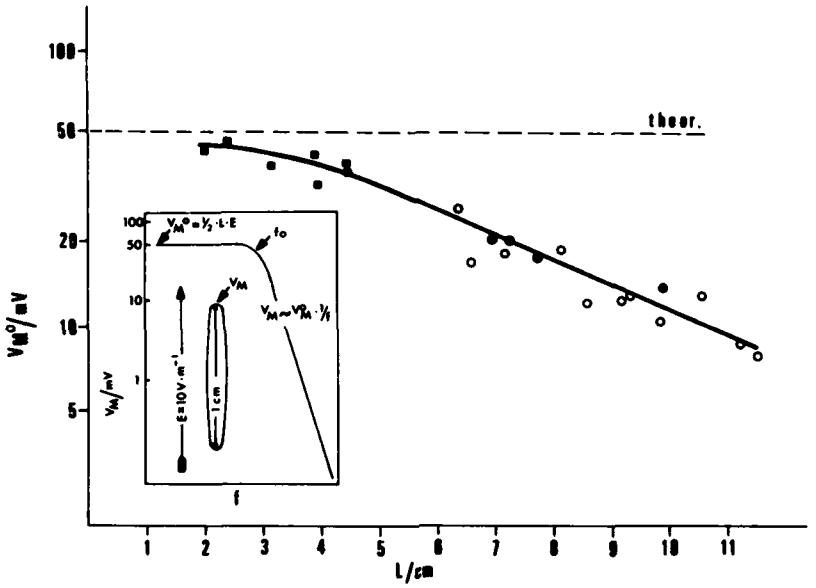

Fig. 4: Field-induced membrane potential differences at plant cells exposed to an alternating electric field. The more the cell length increases the wider becomes the lag behind the theoretically calculated value at the ends of the cells.
In each case E is chosen, whereby $L \cdot E = 100$ mV.

3.2 Hazardous Current Density Curves

As conclusion of chapter 3.1, curve A in Figure 2 can be considered the "envelope" which delimits the experimentally found current density values for stimulating effects. Current densities which are produced by electrodes or which are induced by external electric or magnetic fields may result in a stimulation effect on neurons and muscle cells with values above the plotted curve A. An unexpected stimulation of muscle cells may lead, for instance, to a situation of fright that can trigger a hazard. When, after a considerably long duration of influence by current densities above this "envelope", cerebral nerves in major spheres are stimulated at the same time acute neurologic symptoms cannot be excluded such as may occur in electrical accident, (e.g. increased blood pressure, convulsions in vessels, spasms of the breathing system, paralyses). In this sense, Curve A may be called the "threshold curve for a possible hazard". The higher current density curve C for the extra systole elicitation is employed here as the "threshold curve of injury". Ventricular fibrillation - that may occur above this curve - in the sense of a definition of the injury is considered here to be that event which must be avoided. Curve C is valid for exposure times of about one heart period, i.e., approximately 1 s for man. For shorter exposure times much higher thresholds are necessary (see Fig. 3 and 7). This must be considered when personnel health limits are defined, furthermore, a sufficiently wide safety margin from the injury curve must be selected (see chapter 3.5).

With higher frequencies, the threshold values for stimulating effects are close to current densities that result in a thermal effect. A current density of roughly 1mA/cm² generates a specific absorption rate of 2 W/kg in the tissue (specific conductivity 0.2 S/m), which may result in a rise in temperature by 1 °C in muscle tissue over a period of one hour (without consideration of the thermal transfer by thermal conduction and blood circulation). With a current density of 5 mA/cm² (reached with curve A at 100 kHz) the same heating effect occurs in less than few minutes. The comparison shows that a possible injury occurs as a result of a stimulating effect rather than a thermal effect for a short period of time with frequencies up to the range from 30 to 100 kHz.

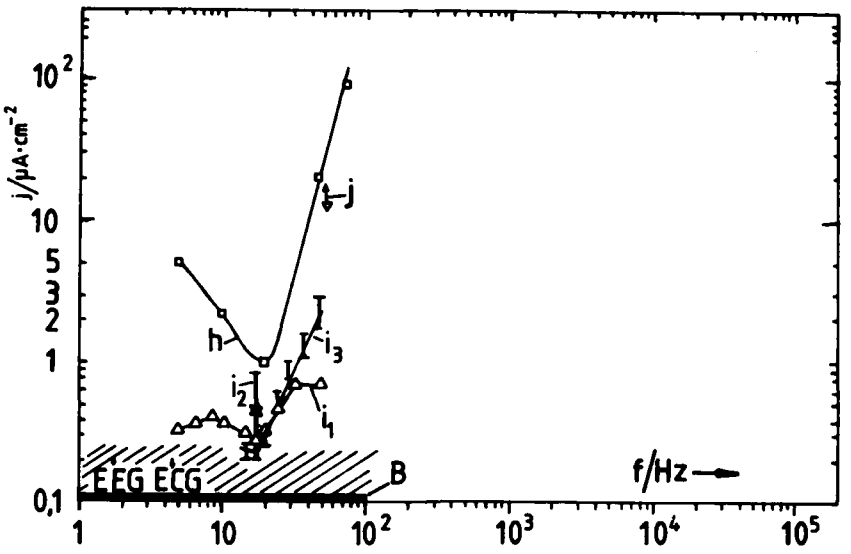

Fig. 5: Calculated current densities for the threshold values of different biological effects,
h: electrophosphenes
i: magnetophosphenes
j: variation of optically generated reaction potentials
B: average value of naturally flowing current density in the brain; the values of the current densities due to the electrical events in the brain or in the heart are usually much larger at a microscopic level.

3.3 Biological Effects with Current Densities below the Stimulation Thresholds

Electro-physiologic studies have shown that information can be transferred between neuronal elements even without action potentials (Schmitt et al., 1976). Minor potential variations by 0.1 mV in one neuron may influence the activity in other neurons by a synaptic effect. For instance, in processes of the retina, obviously small local circuits with synapses play a certain part for information transfer in both directions, before the information is passed from a ganglion cell as an action potential through the optic nerve. Today, the view prevails that in the brain small graded potential variations in the range of 0.1 mV are important in many processes. It must be expected, therefore, that current densities in electrical events in the brain, which are below the stimulation thresholds, may take an influence on functions of the brain. As experimental studies in this direction could so far be hardly carried out an attempt should be made to delimit the range in question on the basis of a small quantity of data only.

3.3.1 Electro- and Magnetophosphenes

The generation of light effects (phosphenes) under the influence of electric currents or magnetic fields have been known for a long time. Lövsund et al. (1980, 1981) localize the mechanism to certain areas on the retina.

Adrian (1977) measured the threshold current intensities for phosphene generation with alternating currents of different frequencies. As one electrode was applied directly at the eye of the test subject Adrian was able to give the minimum current density for the generation of phosphenes. Adrian's data is plotted in the form of curve h in Figure 5. Caused by the electrode application, however, current paths are possible that lead to lower values of the current density in the retina. This would imply a shift of curve h to lower current density values.

We carried out own measurements of the thresholds of magnetophosphenes with 22 volunteers (13 male, 9 female). The head was exposed to a sinusoidal magnetic field of an induction coil (diameter 30 cm, length 30 cm, 400 turns), using an artificial illumination of the room. The magnetic field was switched on and off. The threshold of phosphenes for switching on the field and the dependence on the frequency was measured. Two volunteers did not observe magnetophosphenes with our maximum field strengths (12 mT, 17 Hz). Mean and standard deviation of the other 20 volunteers for 17 to 20 Hz are shown in Fig. 6 (point 0).

Because some volunteers did show little concentration, we analysed the frequency dependence of the phosphene threshold with a subgroup of 10 persons (2 female, 8 male). Mean values and standard deviations of this subgroup are shown in Fig. 6. We observed an optimal frequency of 17 Hz. 2 persons did show threshold values below 2 mT at 17 Hz. We calculated a weighted regression straight line between 18.5 and 50 Hz (Fig. 6). The thresholds in the dark room were about 2 to 3 times higher than those measured with artificial illumination for all volunteers. A correlation of the threshold values with the age of the volunteers was not observed. The values given in Fig. 6 could be transferred into current densities in the peripheral spheres of the head (eye, cerebral cortex) using eq. (8) with a diameter of the current loop of 15 cm and 0.2 S/m for the conductivity (point i_2 and curve i_3 in Figure 5).

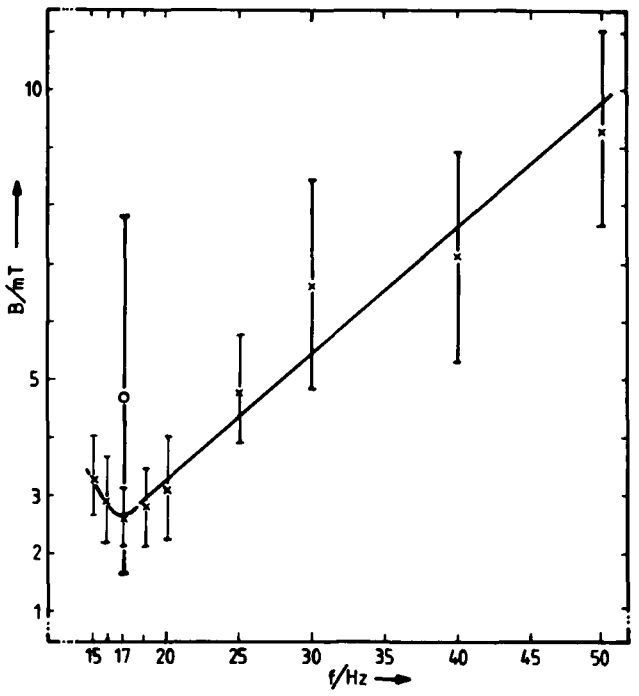

Fig. 6: Frequency dependence of the magnetic induction for the perception of magnetophospenes. (0): mean and standard deviation of the perception thresholds of 20 volunteers. (x): mean and standard deviation of the perception threshold for a subgroup of 10 persons.

Silny (1981) studied the subjective perception of flicker phenomena in volunteers exposed to strong low-frequency magnetic fields. Being generated by a Helmholtz coil arrangement, curve i_1 in Figure 5 is a typical threshold curve obtained by Silny and transformed into current density values. The mean threshold values of Silny, however, are higher by a factor of about 2 to 3 than curve i_1. Similarly, the threshold values of Lövsund et al. (1979) are larger; however, this may be due to another magnetic coupling, because electromagnets were used placed on the temples of the volunteer.

Because of the assumptions for the product $R \cdot \sigma$ in eq. (8) it may not be excluded that the curves i_1 and i_3 of Figure 5 are running at higher values and so coincide almost with the curve h of Adrian. One may conclude, however, that magnetic current densities determined here are below the threshold current density for biological effects of 1 to 3$\mu A/cm^2$ hitherto quoted in literature.

3.3.2 Variation of Reaction Potentials in Low-Frequency Magnetic Fields

Silny (1981) examined test persons to study the influence of 50 Hz magnetic fields on optically generated reaction potentials. A 50 Hz magnetic field with an induction of 60 mT changes the polarity of the observed reaction potential, corresponding to a magnetically induced current density of roughly 14 $\mu A/cm^2$ in the cerebral cortex (j in Figure 5). An important point is the observation that the changed polarity is retained for some time after the field had been switched off; obviously, a time of recovery is required for return to the initial condition. In this context, subjective complaints such as headache must be mentioned, too, which the test subjects suffered.

3.3.3 Field-Induced Potential Differences of 0,1 mV

The idea outlined in chapter 3.1.5 is applied here, but reduced by the factor 100 between 1 and 100 Hz considering now the current density range which must be assigned to field-induced potential differences in the range of 0.1 mV within in this frequency range. If potential difference variations of this magnitude should be the cause of certain biologic effects that have been noted, the corresponding thresholds for the current density ought to come under the magnitude of 1 - 3 $\mu A/cm^2$. The relevance of field-induced potential differences of 0.1 mV, however, needs further and more specific explanation by continued studies and experiments.

3.4 Safe Current Density Curve

One may estimate "safe" field-induced current densities by considering the naturally flowing currents in the brain as a result of the electrical events in the brain. The measured values, which are recorded using extracellular electrodes on the cortex surface (typical values 50 - 100 μV with 1 cm electrode spacing), furnish a current density of roughly 0.1 $\mu A/cm^2$ (Bernhardt, 1979, Childers, 1977). The current densities within the brain may vary strongly at a microscopic level - depending on the cerebral nerve anatomy - and may well be as high as 10 to 100 $\mu A/cm^2$, e.g. on the surface of cells that are electrically active at the time of measurement (Plonsey, 1974). The comparison against the naturally flowing currents, in combination with the frequency range from 1 to 100 Hz, leads to the conclusion that brain

current densities due to external electric or magnetic fields or generated through electrodes should remain without any influence on neurons when the current densities remain below 0.1 μA/cm2 approximately. The same conclusion is valid for the influence of electric fields within the tissue on other mechanisms on the molecular or cellular level, i.e. effects of the induced electric field strengths on biochemical responses, on blood cells, on morphology and cell development should also be excluded for field strength corresponding current densities below 0.1 $\mu A/m^2$.

This conclusion should be valid for the microscopic cellular level. It is clear, that - one more macroscopic level - there is a difference between phase and direction of currents due to external fields on one hand and the naturally flowing currents on the other hand. If biological effects occurring at lower current densities are observed, than other action mechanisms should be responsible than those discussed here.

The limit is indicated by curve B in Figure 5. This curve may be considered to be a "curve of the limits of the safe range". Biologic effects as the consequence of direct action of electric fields onto neurons must be expected above this curve, and their existence has moreover been demonstrated in the range between 5 and 100 Hz.

3.5 Limit Value Curve

In order to define limit value curves, it is possible to take into account a safety margin of 100 from curve C (Fig. 2). The factor 100 can be extracted from IEC-Publication 479 (1983), where this factor is between the range, where the ventricular fibrillation is likely, and the lower range, where usually no reaction effects are observed, assuming a flowing time of 1 second for the body current. In Fig. 7 the time/current zones of physiological effects of ac currents (15 - 100 Hz) on persons are presented,

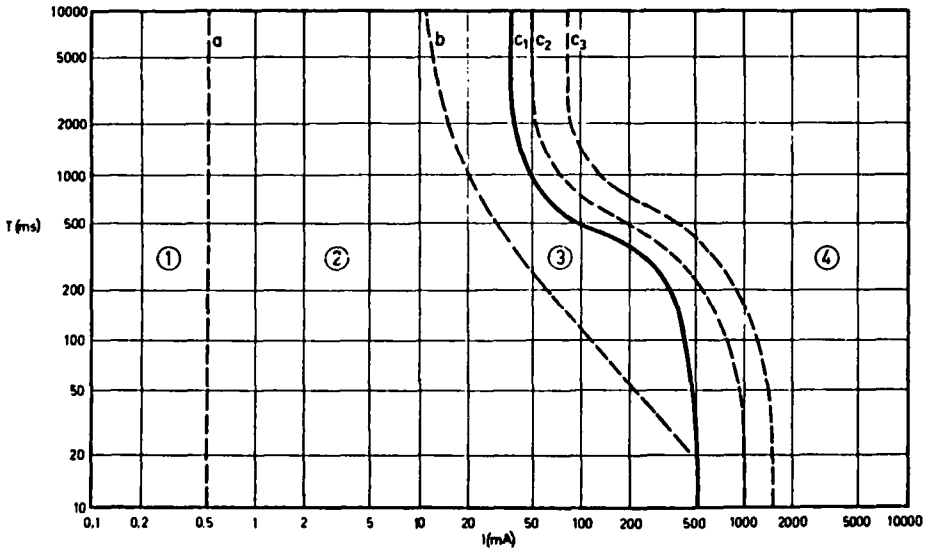

Fig. 7: Time/current zones of effects of ac currents (15 - 100 Hz) on persons. Zone 1: Usually no reaction effects. Zone 2: Usually no harmful physiological effects. Zone 3: Usually no organic damage to be expected. Likelihood of muscular contractions and difficulty in breathing, reversible disturbances of formation and conduction of impulses in the heart, including atrial fibrillation and transient cardiac arrest without ventricular fibrillation increases with current magnitude and time. Zone 4: In addition to the effects of zone 3, probability of ventricular fibrillation increases up to about 5 % (Curve C_2), up to about 50 % (Curve C_3) and above 50 % beyond curve C_3. Increasing with magnitude and time, pathophysiological effects such as cardiac arrest, breathing arrest and heavy burns may occur. (From IEC-Publication 479, 1983).

Referring to Figure 8, where the threshold values of the electric current density for different biological effects of Figure 2 and Figure 5 are summarized, the factor 100 would lead to a current density of about 1 $\mu A/cm^2$. However, there are two reasons that lead to the conclusion that a higher safety margin is necessary. Firstly, as Fig. 8 shows, there are biological effects occurring at lower current densities than 1 $\mu A/m^2$. Secondly, current density values of 1 - 3 $\mu A/cm^2$ may induce miniatur potentials in the order of 0.1 mV.

Here, particular importance must be awarded to the brain as the most important switching and control center of the body. As has been set out, today the view prevails that small potential variations below the depolarization processes required for action potentials have a role in many cerebral functions which is greater than had been assumed so far. Further information is urgently needed in this respect, mainly to demonstrate whether the health condition will be impaired with exposure to current densities in this range for a long period of time. Here, too, the field strength or current density in the brain should be known so that the results gathered in animal experiments can be transferred to the human body. Besides the reasons mentioned already, one should take the following additional points into consideration for the safety margin:

- The lack of sufficient long-term experience;
- The data on conversion of external electric and magnetic fields into current density in the body involves different assumptions and premises which must be balanced with an additional safety factor;

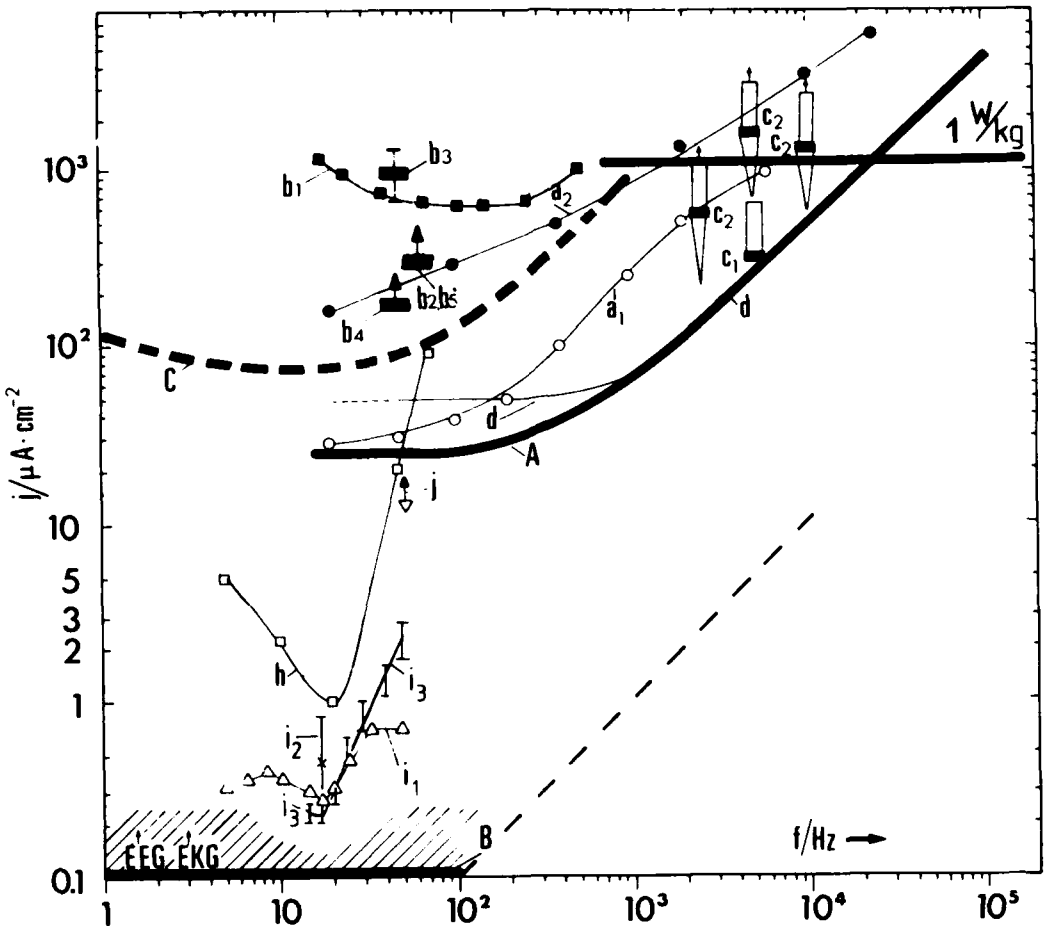

Fig. 8: Threshold values of the electric current density for different biological effects. For the explanation of the symbols see the legends of the Figures 2 and 5.

- As here, human exposition to electric or magnetic fields is discussed there is a substantially higher uncertainty on the precise paths of the field-induced currents than the uncertainty existing with galvanic current supply into the human body.

As a conclusion, curve B in Figure 8 is suggested as a limit value curve in evaluating human exposure to external electric and magnetic fields. However, additional theoretical and empirical studies will have to demonstrate whether this safety factor is open to reduction or whether the factor must be increased. Here, studies on indirect effects of the fields also should be included. It is obvious, that one can assume for curve B a similar increase above 100 Hz like that of curves A and C in Figure 8. This increase of the current density is indicated in Fig. 8 by the dotted line of curve B for frequencies exceeding 100 Hz.

4.0 BODY CURRENT DENSITIES INDUCED BY EXTERNAL LOW FREQUENCY FIELDS

This Chapter quotes the external electric field strengths (E_e) and magnetic field strengths (induction B_e) which, as a function of frequency, induce certain mean electric current densities in the human body. On the one hand, the current densities for the heart and its environment are analyzed, and on the other hand, the human head with the brain as the most important switching and control center of the body will be dealt with as another "critical" organ.

Studies on distribution of the electric field in homogenous spheres (radius R) exposed to a plane electromagnetic wave, show that with frequencies below 10 MHz, approximately, and in biologic material (characterized by ε and σ) the electric field strength in the sphere is composed of an electric term E_i^E and a magnetic term E_i^B (Lin et al., 1973, Bernhardt, 1979):

$$E_i^E = \frac{3\varepsilon_0 \cdot \omega}{\sigma} \cdot E_e = A_k \cdot \frac{f}{\sigma} \cdot E_e \qquad (4)$$

wherein $A_k = 6\pi\varepsilon_0$ and

$$E_i^B = \pi \cdot f \cdot R \cdot B_e \qquad \text{(for sinusoidal fields)}, \qquad (5)$$

or

$$E_i^B = 0.5\ R \cdot dB/dt \qquad \text{(for any time varying field, Budinger, 1979)} \quad (6)$$

Corresponding studies with ellipsoids (Johnson et al., 1975; Durney et al., 1975; Massoudi et al., 1975) have shown that here, too, in a rough approximation, the external electric and magnetic fields may be considered separately and independently of each other.

Elongate spheroids with longitudinal axes parallel to the external E vector must be deemed the "worst case" with external electric fields. For this reason, the electrically induced field strengths in the body can be determined from information on the internal field strengths or on the total of the absorbed power for which values are at hand.

Further information from studies on the distribution of the electric field or the absorbed power in different parts of the human body have shown that, with frequencies below 10 MHz, the internal field strength increases directly proportionally to frequency with a predetermined external electric field strength (Chen & Guru, 1977 a, 1977 b, Gandhi et al., 1979; Hagmann & Gandhi, 1979). This means that the application of a suitable value for A is possible in the relationship between the internal and the external electric field strengths (eq. 4), depending only on the body part or organ considered and, furthermore, on the exposure conditions.

Regarding the magnetically induced current densities, both the cardiac region and the brain are each considered as homogenous spheres, however, of different radius. The electrically and magnetically induced current densities are calculated by

$$j^E = A \cdot f \cdot E_e \quad (7)$$

$$j^B = \pi \cdot R \cdot \sigma \cdot f \cdot B_e \quad (8)$$

(The current densities must be added vectorially when both an electric and a magnetic field is present.)

With suitable values for A and σ, the electric current densities for the head and the cardiac region with external electric and magnetic fields and frequencies below 100 kHz are calculated separately of each other, using the equations (7) and (8) above.

4.1 Electrically Induced Current Densities

The A-values were determined by two different methods. Firstly, calculations of the internal electric field strength or of the power absorbed in so-called block or cell models of the human body were applied. Data from studies of different authors on the absorption in the high-frequency range within the quasi-static range were used. The upper part of Table 1 gives a survey of data originating from different authors and values of the constants A for both the thoraic and brain region. Table 1 shows that the constant A - and thus the current density - in elongate spheroids or in the head of block models is up to 30 times higher than the current density in the simple spherical model.

Apart from the data from studies on the absorption in the high-frequency range, and beside the employment of the extrapolation method within the quasi-static range, A was determined also by way of calculation of the current densities for the head and the thorax on the basis of the field strength measured on the body-surface at 50/60 Hz (Deno, 1979; Kaune & Phillips, 1980; Schneider et al., 1974; Guy & Chou, 1982), see lower part of Table 1. The A-values, determined by entirely different methods, coincide satisfactorily. As the problem here is only the determination of the order of magnitude of the body current densities the same value $A = 3 \cdot 10^{-9}\ S \cdot Hz^{-1} \cdot m^{-1}$ was applied in relation to the cardiac region as well as for the head.

Figure 9 shows the values of the electric current density applying to the cardiac region and to the head, as a function of frequency and the external electric field strength. The curves B and A have been transferred from Figure 8, using equation (7) above, to Figure 9. The ordinate is plotted up to that value which is generally deemed the value of ionization of the air (roughly $3 \cdot 10^6$ V/m).

Additionally, some more data were plotted in Figure 8. Firstly, the shaded area B' is corresponding to current densities of 1 $\mu A/cm^2$ in other parts of the body where the current density may be higher than in chest and head, i.e. in neck and ankles. The shaded area B' should recall the fact that the distribution of the current density within the body is very inhomogeneous and conclusions from the simplified presentation of Figure 9 should be drawn only with care.

Secondly, some more data were plotted into Figure 9, which do not fit into the concept of body field strengths, as outlined in this paper. These data concern the direct perception of electric fields by surface effects (moving hairs) and the perception and annoyance of capacative spark discharges. The following data were selected and plotted in Figure 9:

- response threshold of 1kV/m for 5 % of men with sensations on their raised hands to an electric field produced by an overhead transmission line (IEEE, 1978);
- response threshold of 7 kV/m for 50 % of men with raised hands (IEEE, 1978);
- response threshold of 3 kV/m for 5 % of mean with sensations in their head, head hair, or tingling between body and clothes (hands at sides; IEEE, 1978);
- response threshold of 20 kV/m for 50 % of men with sensations on head, head hair or tingling (IEEE, 1978);
- median perception levels of 3 kV/m for spark discharges to the finger, an umbrella or a person acting as charge collecting object (IEEE, 1978; Zaffanella & Deno, 1978);
- median annoyance level of 7 kV/m for spark discharges to the finger or thumb, an umbrella or a person acting as charge collecting object (IEEE, 1978).

Model	SAR/W kg^{-1}	Ea/Vm^{-1}	f/MHz	σ/Sm^{-1}	A/10^{-9} S Hz^{-1} m^{-1} Heart	Brain	Author
Sphere			< 20		$6\pi\epsilon_0 = 0.17$		(class. solution)
Ellipsoid	$2.5 \cdot 10^{-5}$	61	1	0.6	2.8		Durney, 1975 Massoudi, 1977
Ellipsoid :							
skinny man	$4 \cdot 10^{-5}$	61	1	0.6	3.6		Durney, 1978
average man	$2.5 \cdot 10^{-5}$				2.8		
child, 5 years	$2 \cdot 10^{-5}$				2.5		
fat man	$1.5 \cdot 10^{-5}$				2.2		
child, 1 year	$0.9 \cdot 10^{-5}$				1.7		
cell-model of man	head:	1	30	0.6			Chen, 1977
	$E_i = 0.0612$ V/m					1.2	
	chest:						
	$E_i = 0.116$ V/m				2.3		
	head:						
	1.13 mW/m^3					1.23	
	chest:						
	4.07 mW/m^3				2.33		
cell-model of man in free space	head, mx. value:	61	10	0.6			Gandhi, 1979 Hagmann, 1979
	$3.2 \cdot 10^{-3}$					3.2	
	head, mean value:						
	$1 \cdot 10^{-3}$					1.8	
	chest, center:						
	$3.5 \cdot 10^{-3}$				3.4		
	chest, mean value:						
	$1 \cdot 10^{-3}$				1.8		
cell-model of man on ground	head, max. value:	61	10	0.6			
	$6.2 \cdot 10^{-3}$					4.5	
	head, mean value:						
	$2 \cdot 10^{-3}$					2.5	
	chest, center:						
	$1 \cdot 10^{-2}$				5.7		
	chest, mean value:						
	$3 \cdot 10^{-3}$				3.1		
Man on ground	head:						
	j = 4.8 nA/cm^2 (from field strength at surface)	1000	$5 \cdot 10^{-5}$			0.96	Schneider, 1974
	head:						
	j = 60 nA/cm^2	10^4	$6 \cdot 10^{-5}$			1	Kaune, 1980
	chest:						
	j = 190 nA/cm^2	10^4	$6 \cdot 10^{-5}$		3.2		
	head:						
	j = 13.9 nA/cm^2	10^3	$6 \cdot 10^{-5}$	0.12		2.3	Guy, 1982
	chest:						
	j = 18.5 nA/cm^2	10^3	$6 \cdot 10^{-5}$	0.18	3.1		

Table 1: **Survey of data furnished by various authors and employed in calculation of the current densities induced in the head and in the cardiac regions.**

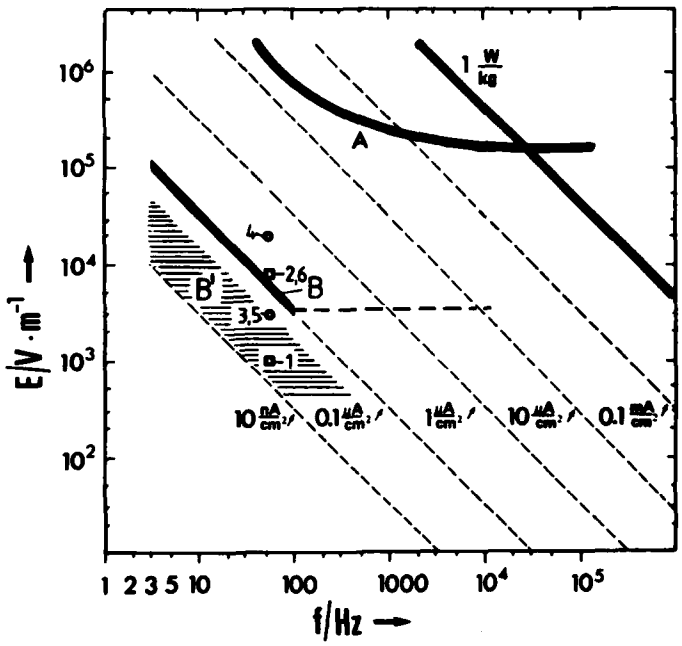

Fig. 9: Electric field strength in man's environment, which induces approximately the indicated current density in the head and in the cardiac region (longitudinal axis of man parallel to orientation of the field; the numerical values given for the field strength apply to the undisturbed field).
Curve A: threshold value curve for stimulating effect;
Curve B: limit curve of the safe range;
Hatched area B': current density of 1µA/cm² in part body regions with higher induced field strengths, i.e., neck or ankles;
1: response threshold for 5 % of men with sensations on their raised hands;
2: response threshold of 50 % of men with sensations on their raised hands;
3: response threshold of 5 % of men with sensations in their head, head hair, or tingling between body and clothes (hands at sides);
4: same effects like (3), however for 50 % of men.
5: median perception levels for capacitive spark discharges to the finger or thumb, an umbrella or a person acting as charge collecting object;
6: median annoyance level for capacitive spark discharges, umbrella or person acting as charge collecting object.

4.2 Magnetically Induced Current Densities

Different authors give different values for the low-frequency conductivity of the myocardial tissue and the nerve tissue. Table 2 gives a survey of various values.

In the model calculations set out here, differences in conductivity of the white and the grey cerebral substance, and the anisotropic nature of conductivity at frequencies below 10 kHz approximately are left out of consideration. Especially with regard to the anisotropic nature of the conductivity, high ratios of transverse to longitudinal impedances were observed. Zheng et al. (1984) reported values between 10 and 20 for the ratio of transverse to longitudinal impedance from studies on skeletal muscles of five mammals and chickens. It should be remembered here that the exact current paths of the magnetically induced currents and the exact magnitudes of the current densities are not known and that only rough approximations can be made.

For the calculations set out here a value of 0.2 S/m has been employed for the specific conductivity of the cerebral substance, whilst a value of 0.25 S/m has been used for the myocardial tissue.

For R in equation (8), a value of 7.5 cm for the head, and a value of 6 cm for the heart was substituted. As a result of the selection of σ, the products $\sigma \cdot R$ are equal for the heart and head, therefore, the current densities in peripheral regions of the heart and of the brain may be presented by a single representation.

Figure 10 represents these current density values as a function of frequency and of the external magnetic field strength (magnetic induction). By application of equation (8), the curves B, A and C have been transferred from Figure 8 into this diagram. The values given for the current densities are applicable only to the peripheral regions of the heart or the head, e.g. in relation to the cerebral cortex, following the definition of equation (8). For zones closer to the center of the heart or the head, higher values for the magnetic induction are necessary to induce the same current densities.

In order to demonstrate the effect of larger effective current loops and conductivities, which may occur in peripheral regions of the heart, the hatched area B' was plotted in Fig. 10, corresponding to a current density of about 1µA/cm². Furthermore, the threshold values for magnetophosphenes are shown in Figure 10 (data from Silny, 1981, and Bernhardt, this paper).

Cardiac muscle σ/S · m^{-1}	Remarks	Nerve Tissue σ/S · m^{-1}	Remarks	Author
0.11 – 0.17	dog, rabbit	0.13 – 0.2	rabbit	Schwan, 1956 Schwan, 1957
		0.18 – 0.22	human	Schwan, 1963
0.13	canine, randomly-oriented muscle average	0.15	rabbit, cat cow, pig average	Geddes/Baker 1967
		1.1	cat, white matter, longitudinal	Nicholson, 1965
		0.13	cat, white matter, transverse	
		0.26	rabbit, cortex, grey matter	Adam/Schwan 1974
		0.36	white matter, longitudinal	
		0.16	white matter, transverse	
0.35	pig	0.25	pig	Schelter, 1983
0.25		0.2		(used in this paper)

Table 2: Survey of data furnished by various authors on the low-frequency conductivity in relation to the tissue of the myocardial and the nerve tissue

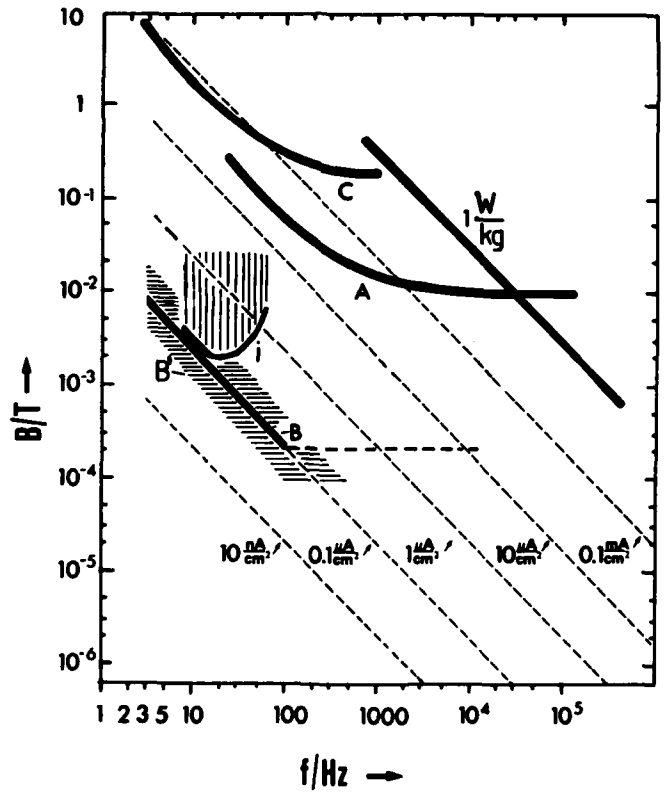

Fig. 10: Magnetic induction in man's environment with sinusoidal variations of the field in relation to peripheral regions of the head and the heart, which induce the indicated current densities.
Curve A: threshold value curve for stimulating effects
Curve B: limit curve of the safe range
Curve C: diastolic stimulation threshold
Hatched area B': current density of 1μA/cm², in peripheral regions of the heart, assuming larger current loops and larger values of the conductivity;
i: threshold values for magnetophosphenes.

5.0 CONCLUSIONS

The field strength/frequency diagrams initially permit a rapid orientation about the respective mean electric current densities that will have to be expected in the human head or in the heart with predetermined external electric or magnetic field strengths as a function of frequency. The diagrams can be likewise applied to the exposure to fields in the power engineering range, and to other frequencies. When the ordinate is used to plot the magnetic induction it should be noted that the ordinate origin must be shifted upward or downward, in accordance with equation (8), for other radii or other conductivity characteristics.

The explanations given in chapter 3 on the relevance of curves B, C and A, are equally valid here by way of analogy. Figure 9 shows that the stimulation threshold curve A can practically not be reached in case of exposure to external electric fields as the required values of the electric field strengths are excessively high. Conditions are different, however, with magnetic fields. Industrial metal smelting and processing requires magnetic induction at levels far above the curve A (Figure 10). According to some measured data, the values required for stimulation of neurons are not reached, however, at working places. With values of the electric or magnetic field strength below curve B, an influence on neurons, on biochemical responses, on blood cells, on cell morphology and cell development is not expected. In the event that biological effects should yet be noted with such fields strengths these effects must be based on action mechanisms other than those so far described. An example may be the direct effect of a static magnetic field on the velocity of a biochemical reaction, the first reaction steps being determined by the rules of the quantum theory.

The statements given here are confirmed for the power engineering range of electric 50 and 60 Hz fields in so far as the extensive laboratory and epidemiologic experiments and studies so far carried through with both animals and test subjects with electric field strengths up to roughly 20 kV/m and with magnetic induction values of 0.3 mT did not reveal any indication of effects involving a health hazard or affection (Bridges et al., 1981; Repacholi, 1984; Schaefer, 1983; Suess, 1981; UNEP, 1984), considering perception of electric fields by surface effects or by spark discharges as harmless.

When the curve B is employed to evaluate human exposure to external electric and magnetic fields or to have a basis of discussions on the definition and determination of personnel health limits, attention must be drawn again to the fact that the Figures 9 and 10 are suited only to give an idea of the magnitude of the current density in the body. Mean values were taken as the basis to determine the distribution of the electric field in the heart and the head, whereas the exact current paths are not known. Local increases of the internal field strength cannot be precluded. The extent of locally excessive field strengths needs further elucidation by continued studies. Safety factors can be defined more precisely only by further studies. Long-term studies with animal experiments and epidemiologic examinations of personnel are particularly important methods.

One second (1 heart period) should be taken as a base for the exposure time for which the field strengths should be averaged. For shorter exposure times higher values of field strength may be accepted. For an exposure of the extremities to magnetic fields special considerations are necessary, leading certainly to higher limiting values for the field strength.

The model described here, however, does not furnish any statement on the extent by which other factors and secondary effects, too, must be employed to arrive at limits values. Examples are surface effects or currents flowing in the body when metal objects are touched in which potentials are induced (burns and micro shocks, Gandhi, et al., 1982 (for field perception and spark discharges see chapter 4.1)), furthermore, the influence of fields on life-saving installations, pacemakers (Bridges & Frazier, 1979) etc. The evaluation of field strength levels, which lead to perceptible, but harmless effects, may be different for the general population and for occupational exposure. It may be possible to eliminate perceptible effects for workers by suitable technical measures or to inform them of the secondary effects.

Levels of exposure of the general populations should be limited to values low enough to avoid perceptible effects even if harmless or the perceptible effects should be excluded by technical measures - at least for the dwellings where a continuous exposure of persons cannot be excluded.

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HAZARDS OF VLF ELECTROMAGNETIC FIELDS

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SUMMARY

Biological hazards to humans exposed to VLF electromagnetic fields may result from electric shock, spark discharge, elevation of tissue temperature and burns. A number of nonthermal-nonshock effects, such as the neurasthenic syndrome had been reported widely in the Soviet and East-European literature. Generally a subject exposed to even the highest electromagnetic fields in the environment will not experience an electric shock unless he comes in contact with the ground or objects such as vehicles, crane cables and guy wires. The steady state current from touching objects increases directly with the frequency. On the other hand, the physiological shock effects of the current are less pronounced with increasing frequency. However, at frequencies where the shock hazard is negligible, deleterious heating of the body occurs for whole body average specific absorption rates (SAR) greater than 4 W/kg and localized values of SAR greater than 80 W/kg so VLF exposure levels should be limited to prevent SAR from reaching one tenth of these levels. Free space exposures to fields of less than 1 kV/m electric field strength should be safe if proper precautions are taken to limit direct body contact with conducting objects.

Symbol List:

FM	frequency modulation	mJ	millijoules
Hz	Hertz	mW/cm2	milliwatts per square centimeter
kHz	kilohertz	V/m	volts per meter
kV/m	kilovolts per meter	A/m	amperes per meter
mA	milliampere	W/kg	watts per kilogram

INTRODUCTION

Over the past decade there has been considerable focus of attention by the Radio Frequency Radiation (RFR) health and research community on the biological effects of RFR and on establishment of safe exposure standards for the RFR band of 10 MHz through 10 GHz. This attention was stimulated by the rapid proliferation of new RFR emitters, including FM and TV stations, microwave ovens, microwave communication antennas, and military and civilian radar systems. At the same time with newly proposed high power extra-low frequency sources, such as ultra high voltage (UHV) power transmission lines and switch yards, as well as communications systems such as project ELF, attention was focused on frequencies below 100 Hz.

In comparison to construction of new UHV power transmission and microwave facilities, there has been relatively little new construction during this period of RFR sources in the very low (VLF), low (LF), and medium frequency (MF) bands spanning the 3 kHz-3 MHz frequency range. Therefore, little attention was given to assessment and development of safety criteria for these frequencies. In addition, a scientific consensus in the Western countries seems to indicate that there should be no serious health problems other than electric shock and RF burns associated with the use of high power sources operating in the VLF-MF bands. Energy coupling to the human body even under the high field exposure levels encountered in practice would be far below that commonly experienced for exposures at microwave frequencies. Recently, however, increased attention has been directed toward possible problems and the lack of safety standards associated with frequencies in the 10 kHz - 3 MHz band.

International standards pertaining to the VLF-MF frequency range are shown in Table 1. The top two-thirds of the table covers the United States and NATO standards while the lower one-third covers the Soviet Union and some East European countries. The North Atlantic Treaty Organization (1) has set a new standard, 265 mW/cm^2 (equivalent electric field strength E of 1000 V/m, and magnetic field strength of H of 2.65 A/m) from 10 kHz - 1 MHz.

In the development of the new American National Standards Institute (ANSI) C95.1 - 1982 Radio Frequency Protection Guide (RFPG) (2) the ANSI C95.4 Subcommittee was faced with a problem of where to place the lower frequency limit. On one hand the frequency squared dependence of absorbed energy in exposed bodies indicated that the standard could be allowed to increase by the inverse square of frequency. On the other hand, if the magnitude of the exposure fields is allowed to increase without limit, other problems such as corona and electric shock effects become pronounced. The ANSI Subcommittee handled the problem by limiting the maximum power density to 100 mW/cm^2 (E = 614 V/m, H = 1.63 A/m). The Subcommittee was unwilling to apply this recommended value, however, to frequencies lower than 300 kHz since it was felt that it would be too conservative as an exposure standard. But the other hazards such as electric shock

could become more of a consideration, requiring a different scientific approach than that taken. The ANSI RFPG is based on a large consensus of U.S. scientists assessing the scientific literature as to what constitutes safe exposure levels. However, the new ANSI RFPG is lower than the old guide by a factor of 10 (1 mW/cm^2 in the 30 to 300 MHz frequency range), since plane wave energy couples more efficiently to the body in that portion of the spectrum. On the other hand, below 30 MHz the energy coupling factor decreases as a square of the frequency and the internal electric field and current decreases directly with frequency, so the guide is allowed to increase by the inverse of the factor, reaching 100 mW/cm^2 (E = 614 V/m, H = 1.63 A/m) at 3 MHz. There is no reason the guide could not continue to increase for free field exposure conditions, but the guide recommends that the power density level of 100 mW/cm^2 and the related field strengths not be exceeded at lower frequencies due to the increasing possibilities of shock and electric discharges if exposed persons contact other objects in the field. Since such a fixed exposure level becomes increasingly conservative with decreasing frequency in terms of simple whole body exposure, the guide does not make recommendations below 300 kHz. If the shock hazard is prevented, however, the basis of the ANSI standard at other frequencies would allow the guide to increase with frequencies below 300 kHz to levels as high as 18,400 V/m at 10 kHz without an increase in absorbed energy or body current above that due to 614 V/m at 300 kHz [(1) to (2) in Fig. 1]. In fact, typical 60 Hz electric field exposure levels of 10,000 V/m on highways crossing under high voltage lines produce negligible current flow in an exposed human [(4) in Fig. 1].

The American Conference of Governmental Industrial Hygienists (ACGIH) (3) on the other hand, was conservative in extending the new ANSI guide of 614 V/m from 300 kHz down to 10 kHz for their standard [(3) in Fig. 1]. Based on exposure hazards due to energy absorption only (discounting the shock hazard from touching nearby large objects), the ACGIH's 10 kHz guide is 900 times more conservative than the 300 kHz ANSI guide. Clearly such a guide will have an impact on the operation of some VLF stations based on typically measured field strength levels of up to 1000 V/m in parking areas near the antenna feed lines.

Other United States standards for the general population found in the middle of Table 1 are somewhat reduced from the NATO, ANSI, and ACGIH standards, with the standards set by Portland, Oregon, at 0.5 mW/cm^2 (43 V/m covering the frequency range 100 kHz - 3 MHz) the most conservative. The Commonwealth of Massachusetts (4) has adopted the ANSI standard for occupational exposures, with a one-fifth reduction to 20 mW/cm^2 or 275 V/m for the general population in the frequency range of 300 kHz to 3 MHz. Since the averaging time for the general population was increased to 30 minutes, the standard still allows short-term exposures of 6 minutes or less which is the same as the ANSI standard. Recently Multnomah County, Oregon (5), adopted the Massachusetts standard for the general population as a result of increasing public concern about the concentration of broadcast station antennas in residential areas and New Jersey adopted the ANSI standard for both the working and the general population.

The most conservative safety standards in the world are those of the USSR and Eastern Europe. The Soviets do not have a standard for 10 kHz, but they do for 50 Hz and for 60 kHz and above. The Soviet standard of 5000 V/m for 50 Hz exposure during an 8 hour working day is very conservative from the standpoint of whole body exposure. It is based on the prevention of any unpleasant sensation of electric spark discharges when exposed humans touch grounded objects in the field (6). The Soviet standard for maximum human exposure is for frequencies between 60 kHz and 3 MHz based on laboratory work showing a threshold level of exposure for biological effects. According to Soviet standard setting philosophy, the Soviet occupational standard for 8 hours/day exposure of 50 V/m electric field strength and 5 A/m magnetic field strength is four to five times lower than the thresholds for effects found in their laboratories (7). On the other hand, for 24 hour/day general population exposure, the USSR standard is 20 V/m at 60 kHz, which is 10 to 12.5 times below their reported threshold levels for effects. Though no Soviet standards exist for frequencies between 50 Hz and 60 kHz, in order to recommend maximum exposure levels in the VLF range where powerline harmonics exist, Filipov and Morozov (6), suggested a direct linear extrapolation between the standards existing at 50 Hz and 60 kHz with maximum exposure electric field strength, E, given by $E = 0.083 (6 \times 10^4 - f) + 20$ where f is the frequency in Hz [(5) in Fig. 1]. Thus according to Filipov and Morozov's equation, the maximum electric field exposure level to 10 kHz would be 4200 V/m. However, if one were to limit body currents due to free field 10 kHz exposure to the same levels allowed by the Soviet 50 Hz standard, one would have to extrapolate [as shown by (6) in Fig. 1], limiting exposure fields to 25 V/m at 10 kHz. This is unrealistic, however, since the neuromuscular system is less sensitive by a factor of 6 to 10 kHz currents than it is to 50 Hz currents (8). Accounting for the decreased sensitivity and the extrapolation, the equivalent 10 kHz exposure would be 150 V/m. If one were using the Soviet standards as a basis for extrapolation, a direct extrapolation from the Soviet 60 kHz exposure standard is preferable, since in contrast to the 200 times increase in frequency based on a 50 Hz extrapolation, only a 6 times decrease in frequency is required. Thus, an extrapolation from 60 kHz occupational exposure level of 50 V/m would yield a maximum exposure level of 300 V/m at 10 kHz, and extrapolation from the general population standard of 20 V/m would yield a maximum exposure level of 120 V/m at 10 kHz.

Since the energy coupling to the body by normal free field or ground plane is negligible even for the highest electric field in the VLF-MF bands, it appears from Western research that exposure levels higher than those in the Soviet Standards could be set. However, one must consider other aspects such as shock or burn hazard when an

exposed person insulated from ground touches a grounded object or an exposed grounded person touches an ungrounded object. The National Electric Safety Code (9) specifies that transmission-line clearance shall be adequate to provide a maximum induced current of 5 mA when the largest anticipated vehicle is short-circuited to ground. Furthermore, the code specifies a maximum of 0.5 mA leakage current from portable electrical tools and household appliances and 0.75 mA for permanently fixed appliances. The maximum current at high frequencies allowed in this code could be increased, since the sensitivity of the neuromuscular system decreases with increasing frequency. For example, at 10 kHz a 35 mA current would produce physiological sensation equivalent to that produced by 5 mA at 60 Hz.

BIOLOGICAL HAZARDS OF VLF-MF ELECTROMAGNETIC FIELDS

Biological hazards to humans exposured to VLF-MF electromagnetic fields may result from any of the following: a) electric shock, b) spark discharge, c) elevation of tissue temperature, d) burns, e) pacemaker interference. A number of nonthermal-nonshock effects, such as neurasthenic syndrome which has been reported widely in the Soviet and East European literature.

The first four of the above interactions are well understood and quantified extensively in the literature but considerable controversey stems from the latter effects.

Electric Shock and Spark Discharges

Generally a subject exposed to even the highest electromagnetic fields in the environment will not experience an electric shock unless he or she comes in contact with the ground, or with ungrounded objects, such as vehicles and guy wires. Dalziel et al. (10) and Dalziel, (8, 11, 12, 13, 14, and 15) have conducted extensive research in quantifying the levels of electric current that produce various physiological effects on humans, including men, women, and children. In addition, such work has been reviewed by Keesey and Letcher (16) and Bernhardt (17). A summary of electric current effects on humans is shown in Table 2 for various degrees of interaction ranging from no sensation in the hand to severe shock. The human organism is most sensitive to 60 Hz current with no sensation up to 0.4 mA and severe shock occurring between 15 and 23 mA. The thresholds increase substantially with increasing frequency. It may be noted from Table 2 that threshold of perception is approximately 10 times greater and the threshold for severe shock is 4 times greater, at 10 kHz than at 60 Hz. The threshold for women is approximately two-thirds that of men and it is estimated that the threshold for children is about one-half of that for men. Shocks that occur without loss of muscle control are generally classified as secondary shocks with little danger of physiological harm. However, with increasing levels of steady-state intensity of the current, muscle control becomes more difficult so that it may not be possible to release a grip on an object. This is considered the let-go current. The minimum let-go values shown in Table 2 are the lower bounds for 95.5% of the population. These values are 9 mA for men, 6 mA for women, and a more conservative figure of 5 mA for children. If the current is increased beyond the value of let-go, there is a possibility that ventricular fibrillation can occur.

The common concern for people working under the electrostatic fields of high power transmission lines is the spark discharge. Such spark discharges occur when two bodies of different potential come in contact. The value of the current depends on the body resistance. If the gap remains between the two bodies, the discharge can occur repeatedly. The threshold energy of spark discharges for perception is about 0.12 mJ, and painful shock occurs at spark discharge energy of 0.5 to 1.5 mJ. Discharge energy depends on the open circuit voltage and the object to ground capacitance and is discussed extensively in the literature (18, 19).

The steady state current from touching objects increases directly with the frequency. On the other hand, the physiological effects of the current are less pronounced with increasing frequency. The National Electrical Safety Code (9) sets maximum 60 Hz current levels for portable and fixed electrical appliances and vehicles under overhead transmission lines. These values are given in Table 3 along with estimated equivalent values at higher frequencies. From the table it can be seen that 27 mA should be the largest body current allowed from touching vehicles near 10 kHz VLF stations. Such a level was used for the design criteria in protecting motorists on the H-3 Hawaii defense highway proposed to pass under an Omega VLF antenna.

Whole Body or Partial Body Heating

Deleterious heating of the body would require whole body average specific absorption rates (SAR) greater than 4 W/kg and localized values of SAR greater than 50 W/kg; the latter corresponding to routine diathermy treatments. The criterion for the ANSI standard at higher frequencies uses a maximum whole body average of 0.4 W/kg and would certainly be a conservatively low level that would not only prevent any heating effects, but, according to the ANSI Subcommittee, would also prevent any other type of biological harm due to effects other than shock. The ANSI rationale is also based on the assumption that the maximum SAR may reach levels as high as 8 W/kg for partial body exposures under the recommended guidelines. A VLF-MF safety guide aimed at simultaneously preventing exposure to levels that can cause shock hazard and exceeding the 0.4 W/kg average and 8 W/kg peak SAR should at least be based on the extensive knowledge that resulted in both the National Electrical Safety Code and the ANSI

Radio-Frequency Protection Guide Criteria.

Other Biological Effects of VLF-MF Electromagnetic Fields

A summary of the results from publications reporting biological effects of VLF - MF fields in the western literature are given in Table 4. Most of the studies summarized in the table where performed on animals or cells. The exposure intensity parameters were expressed differently by the various authors of these publications. These differences make comparisons between experiments and extrapolation to humans difficult. Where possible a rough estimate of the maximum SAR in W/kg was calculated according to the current density in tissue or the Radio Frequency Dosimetry Handbook (20). Except for the direct contact experiments of Geddes et al. (21) and Straub (22), all other SARs are quite small due to the small energy coupling at low frequencies.

In contrast to the western literature, more than half of the Soviet studies were on human workers. They include work by Osipov (23, 24, 25), Machabeli, et al. (26), Volfovskaya, et al. (27), Stefonov, et al. (28, 29) and Kharchov (30). The Soviet researchers also reported effects in animals exposed to VLF-MF fields. Results were reported by Chirkov (31), Chukhlovin and Kotova (32), Kulin (33), Mishchenko and Karamyshev (34), Fukalova, et al. (35), Kolodub and Yevtushenko, et al. (36, 37, 38 and 39). Syndromes of the nervous system and the cardiovascular system were the most frequently observed abnormalities. The reported effects in animals do not seem to be consistent with those seen in humans in terms for the same calculated average SAR.

Discussion

The literature on biological effects of VLF to MF electromagnetic fields indicates that the current status of research is very similar to that of microwave research in the early 1960s. First, there is great inconsistency between the Soviet and Western literatures. The Soviet scientists are further ahead in research activity, especially in hygienic studies. However, their reporting lacks detail. The Soviet papers provide little information on the details of the experiment. In several cases, even the field intensity and frequency are not specified. Detailed methodology of both human and animal studies is not described and therefore the results are difficult to evaluate. Second, there is a major deficiency in the area of dosimetry in both Soviet and Western reports. Biological effects of the VLF and MF electromagnetic fields are related to the amount of current, field strength, or energy in the biological objects. The distribution of current, field, or energy in the exposed objects is determined by the frequency, intensity of the field, and the dielectric properties, size, and geometry of the exposed tissue, as well as by the exposure conditions. The reporting of only the frequency and field intensity of the electric or magnetic field does not give enough dosimetry information about the exposure. Therefore, it is difficult to compare the experimental results from various laboratories. Extrapolation from animal research to humans can not be done without the essential dosimetric information. It would be ideal to be able to estimate the induced current density, electric field intensity, or SAR in tissues for all the reported data. However, it is impossible to give a close estimate because of the lack of essential information on the exposure conditions in most of the papers.

Soviet human studies indicate that the central nervous system, the autonomic nervous system, and the cardiovascular system are affected by VLF - MF radiation. The effects are very similar to those reported for the microwave exposure. There is no Western research to confirm the Soviet findings.

DOSIMETRY

It is now widely recognized by researchers that knowledge of the amount of energy absorbed by the tissues of subjects exposed to electromagnetic fields is essential for analyzing the biological effect on the organism. However, safe exposure standards must be developed and enforced based on the magnitude of the exposure fields. Establishment of the relationship between the exposure fields and the fields or absorbed energy in the body is a fundamental requirement for the setting of exposure standards. Though it has been customary to quantify or express the internal absorbed energy in terms of specific absorption rate with units of watts per kilogram, other quantities may become more important in the VLF-MF band. Exposures in the range of 10 kHz through 3 MHz, the subject of this report, result in relatively low amounts of absorbed energy for exposures of the human body to even the highest fields in the environment. Though the absorbed energies are relatively small, direct physiological effects on the neuromuscular system can result from electric shock and tissue may be damaged locally from electric contact between the subject and metallic objects in the field. Thus, in addition to SAR, other important quantities include electric current density, J, and total current, I, in the body resulting from exposure while in contact with objects or surfaces in free space. Fortunately, since most exposed objects or subjects are small compared to a wavelength of VLF-MF frequencies, the analysis of energy coupling to body and the internal current distributions is considerably simplified since they are amenable to quasi-static analysis. Also, the analysis of field to subject interaction in this frequency range is greatly simplified since work done at one frequency within the range is directly applicable to all frequencies within the range.

Thus one can fall back on the considerable amount of theoretical and experimental work done for the 50-60 Hz frequency range which is directly applicable to dosimetry problems in the VLF-MF range. The reason for this was well illustrated by Kaune and

Gillis (40). They showed, like many others before them, that simplification of Maxwell's equations to cover the quasi-static case shows that the field induced within the body of a living subject over the VLF-MF frequency range is very small compared to the external field. It can be concluded that the perturbed electric field outside of the body and the induced charge density on the surface of the body are independent of the electrical characteristics of the body tissues. The external fields and the induced surface charge can easily be measured by using conducting hollow or solid scale models of animals and humans. The time-varying surface charge on the body induced by the time-varying electric field will produce time-varying currents within the body. Though the distribution of the currents depends on the electrical characteristics of tissue, it has been shown by Kaune and Gillis (40) and Deno (41), that the total conduction current crossing any section through the body is independent of the tissue characteristics. This, in turn, implies that the total induced volume charge in the body is negligible compared to the total induced surface charge. Also, the induced current in the body will be directly proportional to frequency. Though a number of theoretical approaches have been used to quantify the induced currents and energy absorption in living subjects exposed to VLF-MF fields, the experimental approaches have proven to be more precise and most frequently relied upon.

Well-quantified measurements by Deno (41) of currents induced in human bodies by high voltage transmission line electric fields form the most complete and accurate basis for analysing field interactions with the human body. Deno measured body currents in hollow metal mannequins exposed to 60 Hz high electric fields and developed empirical equations for predicting total body current in various parts of the body, such as the neck, torso, waist, perinium, knees, and feet. He also did measurements for various positions in relation to transmission towers, for various distances from the ground, and with hands by the side, outstretched, or overhead. He also measured the unperturbed field enhancement at various locations in the body of the mannequin. Deno and Zaffanella (42) wrote an excellent tutorial on electrostatic and electromagnetic effects of AC transmission lines characterizing the electric induction in objects in proximity with high voltage conductors energized at power frequencies. He provided induced short circuit current formulae and Tables of capacitance for a large number of objects exposed to ELF fields. The objects can be used to simulate various vehicles, objects, humans, and animals in order to quantify body currents and spark discharges resulting from contact with the ground or objects exposed to the fields. Deno also supplied extensive tables showing the statistical impedance, open circuit voltages, short circuit currents, person to vehicle currents, and person to vehicle voltages in terms of cumulative probability for a number of different exposure conditions.

Frazier et al. (43) and Bridges and Frazier (44) measured implanted pacemaker susceptibility to power line frequency exposure through research on animals and man. They developed an expression for the current and voltage distribution in man to allow the quantitation of the exposure levels that cause interference with pacemakers based on semi-empirical models. Expressions were developed to express the linear resistance of the thorax. The following section combines new measurements made by the authors with most quantitative approaches discussed above to aid in the definition of biological hazards peculiar to VLF-MF exposures.

The analysis in this report is directed toward evaluation of worst case VLF-MF electromagnetic field exposures. It can be demonstrated that maximum energy coupling to the exposed body occurs when it is oriented parallel to the electric field vector. Furthermore, for plane waves and most VLF-MF antenna sources, the absorption from the magnetic field is more than an order of magnitude less than that from the electric field for this exposure orientation. Also, most shock hazards from objects in the field are due to the electric component. Thus the following analysis considers only the electric field hazards.

This section discusses the evaluation of body current, current density, potential distribution, resistance per unit length, SAR for each body member, average SAR for the whole body, and conductivity of various parts of the body based on its exposure to VLF-MF electric fields under a wide range of conditions. These conditions include exposure of the subject in free space, with feet grounded, with hand grounded, and with hand in contact with various types of vehicles and wires. The Radio Frequency Dosimetry Handbooks, (20, 45), provide estimates of whole body SAR based on ellipsoidal and prolate spheroid models of man. The handbooks do not provide other information, however, such as current density and localized SAR necessary for a VLF-MF hazard analysis. In this analysis, a more applicable model based on actual measurements on living subjects and true-form phantom models exposed to VLF-MF fields is used to develop computer algorithms for calculating the necessary dosimetry parameters discussed above.

Extrapolation from 60 Hz to VLF-MF Band

Since quasi-static analysis techniques apply to bodies exposed to VLF-MF fields, we may express the total charge on an object close to ground by the expression

$$q = EhC_{og} \qquad (1)$$

where E is the unperturbed electric field strength at the ground, h is the effective height of the object, Eh is the potential of the object, and C_{og} is the capacitance of the object to ground. The current induced by quasi-static field on the object is a

short circuit current I_{sc} that can be measured between the object and ground given by:

$$I_{sc} = j\omega q \tag{2}$$

$$I_{sc} = j\omega EhC_{og} \tag{3}$$

which can be expressed as

$$I_{sc} = j\omega \epsilon_o ES \tag{4}$$

where $j\omega\epsilon_o E$ is the free space Maxwell's displacement current per unit area, $\omega = 2\pi f$, and

$$S = hC_{og}/\epsilon_o$$

is the equivalent area. The equivalent charge collecting area of the human is (pi)(h)(tan(37.7^o). The evaluation of equation (4) based on this equivalent area is:

$$I_{sc} = 0.09h^2Ef \tag{5}$$

where h is the height in meters, E is the electric field strength in kV/m, I_{sc} is the short circuit current expressed in mA, and f is the frequency in kHz.

Deno (41), was able to measure this short circuit current and distribution of the induced currents in the body using a metalized mannequin consisting of insulated material covered with copper foil. The currents in various sections of the body were measured using breaks in the copper foil. Based on the above equations, the induced current is proportional to the charge collecting equivalent area of the mannequin times the uniform free space Maxwell's displacement current per unit area. Deno measured the current for a number of different exposure conditions. We will consider three of these conditions in this analysis.

The first condition is the man exposed with his feet in electrical contact with the ground such that all of the short circuit current flows through the legs to ground. The relative distribution obtained by Deno for this condition is shown in Figure 2a, where the maximum current at the feet is equal to the short circuit current given by equation (5). For exposure well above the ground the maximum current is given by

$$I_{max} = 0.041\ h^2Ef \tag{6}$$

with the distribution shown in Figure 2b. Deno did not measure the current distribution in the arm but only reported the magnitude as 14% of the short circuit level. For this analysis we assume a cosine variation for the arm. Deno also obtained the current distribution for man exposed in close proximity with his feet insulated from the ground but with his right hand grounded. This distribution is shown in Figure 2c with the maximum current (in the arm) given by

$$I_{max} = 0.1\ h^2Ef \tag{7}$$

The actual current distribution within the body for the three exposure conditions expressed above is a function of the internal electrical conductivity of the various portions of the body.

Frazier et al. (43), however, showed through studies on baboons that internal body potentials closely match the external potentials and for actual current flows, equal potential planes were perpendicular to the axis of the body. Assuming this type of current flow, Kaune (46) calculated the current density passing through various portions of the body for 60 Hz. If one assumes a linear frequency dependence for current, the current densities through various parts of the body at any frequency can be predicted. The maximum current density occurs for the minimum cross sectional area of the body which corresponds to the ankles. The value for a 10 kHz - 1 kV/m exposure would be 33 uA/cm^2 based on Kaune's (46) results.

This paper also analyzes the situation where a person may come in contact with an object such as a vehicle, a crane, or a wire in the VLF-MF electric fields. Predicted short circuit current based on calculations of the effective area of such objects may be obtained from Deno, (41) Zaffenela and Deno (47), and The Transmission Reference Book, 345 kV and Above (18). Short circuit currents for exposed objects as a given function of frequency, f, is given in Table 5. The worst case energy coupling to a human in contact with such an object would be based on the short circuit current given in the table,

Since the work discussed above was based on measurements done at 60 Hz, it is

desirable to prove the applicability of the equations and concepts to the VLF-MF frequency range. Thus measurements were made of the short circuit body current, short circuit vehicle current, and person to vehicle current for exposures to various VLF-MF frequencies.

Measurements at VLF Stations

Measurements were done on the Island of Oahu, Hawaii, and near Arlington, Washington in the vicinity of very powerful VLF and LF sources. Sensors were designed and fabricated for measuring the exposure electric fields and short circuit body currents of persons exposed to the VLF sources. Figure 3 illustrates the design and the configuration of the sensors. Electric field sensors are based on the amount of displacement current intercepted by a known area. The sensor consisted of a double-back circuit board with an annular ring cut to isolate a 4 inch diameter disk from the rest of the conducting top surface. The relationship between the charging current grounded through the input of the operational amplifier and the electric field is given by equation (4) where S is the area of the 4 inch diameter disk and E is the electric field strength. Through the use of a capacitive feed back loop on the amplifier the output voltage will be given by

$$V_o = I_{sc}/\omega C = \epsilon_o ES/C \qquad (8)$$

The capacitor C was adjusted so that the operational amplifier output was 1 volt when a current was applied to the disk that corresponded to the displacement current produced by 1 kV/m electric field strength. The output of the device was coupled by an RG 58 coaxial cable to a Fluke 8060A multimeter capable of providing accurate true rms voltage readings up to 200 kHz. The microprocessor controlled meter was also able to operate in a frequency counter mode so that the frequency of the applied signal could be easily determined. The operational amplifier and two 9 volt power supply batteries were shielded by means of a metal case with a removable bottom cover.

The body current sensor consisted of a 9 x 12 inch double-back circuit board with the upper plate connected to the center conductor and the lower plate connected to the outer conductor of the BNC cable. The short circuit body current could be measured simply by directing the current flow from the upper plate to the bottom plate through the multi-meter with the exposed person standing on the double-backed circuit board. Since at VLF frequencies the source impedance of the body acting as a receiving antenna is high compared to the resistance between the lower plate and the ground, no grounding stake was required. The sensitivity and dynamic range of the multimeter was such that field strengths from a few 10ths of volts per meter to several thousand volts per meter could be measured. The meter could accurately sense VLF currents from a few uA to 200 mA.

Short circuit body currents were first measured for exposure to the Haiku, Oahu, Hawaii 10.2 to 14 kHz Omega antenna. Maximum electric field strength levels up to 1 kV/m were found in the parking lot under the RF feed cage adjacent to the transmitting building. The input power to the antenna was 130 kW. Measurements were also made on the West side of the Island under the Lualualei Navy communications station antennas This station operated at a frequency of 23.4 kHz with one million watts input to the antennas. Measurements were made in close proximity to the base of the 1500 foot West tower where the electric field strength varied from 1 to 2 kV/m depending on position. Additional measurements of body short circuit current fields were made in low-frequency fields in the vicinity of the 146.8 kHz Lualualei LF source where field strengths were approximately 76 V/m. The field strengths measured with the sensor shown in Figure 3 agreed very well with field strengths measured with a commercial meter of another design which has been used frequently for surveying the fields of a large number of VLF stations.

The body current measurements, tabulated in Table 6 and graphed in Figure 4 , agree very well with the theoretical equation (5) and measurements previously made by Richard Tell of EPA for persons exposed in the vicinity of Las Vegas broadcast stations. The results clearly illustrate the applicability of 60 Hz experimental work to the VLF-MF dosimetry analysis. The short circuit in person to vehicle current for a number of vehicles was also measured in the vicinity of the Hawaiian and Jim Creek VLF stations. The results of these measurements are shown in Table 7. Note that there is good agreement between measured and the calculated theoretical values.

Measurement of Body Potential and Dimensions

In order to make accurate calculations of the SAR distributions from Deno's (41) body current distributions for different exposure conditions it was necessary to determine the conductivity and the resistance per unit length along the axis of the body and limbs. At VLF frequencies this can be done quite simply by passing a known harmless low level VLF current through the body while measuring the potential distribution at various points. Frazier et al. (43) and Bridges and Frazier (44) demonstrated that for exposure of baboons to 60 Hz with the long axis of the body parallel to the electric field equal potential planes existed perpendicular to the axis of the body. Thus measurements of surface potential accurately predicted the internal potentials required for determining the susceptibility of pacemakers to interference from powerline frequency exposures.

The author of this paper conducted an experiment to determine the potential distribution along the long axis of his body and limbs. The measurements were based on the assumption that the conduction currents were large compared to displacement currents, so impedance at various locations could be characterized in terms of simple resistance measurements. The measurement equipment is schematically shown in Figure 5. A Hewlett Packard signal generator was used to drive an amplifier isolated from the subject by a step-up transformer as shown in the figure. A constant current of 10 mA between 10 kHz and 200 kHz was applied to the body. In order to prevent shock, the current was reduced to 3 mA for the 60 Hz measurements. The electrode applied to the feet consisted of a 9 x 12 inch copper plate and the electrode applied at the head consisted of a four inch square of Ringer's solution-soaked gauze sandwiched between the head and a four inch square of aluminum foil. The voltages and currents were measured by means of the same Fluke 8060A multimeter that was used for the Hawaiian VLF current measurements. The results of the measurements are shown in Figure 5. The 60 Hz measured current values were increased to correspond to a 10 mA body current for consistency with the other measurements.

As a further aid to the detailed analysis of the body current and SAR distribution, the circumference and maximum dimensions of the subject's body and limbs were measured as a function of the position every 5 cm from the feet to the head. An elliptical cross section perpendicular to the axis of the body and limbs was assumed for calculating the minimum dimension and the cross sectional area. As a check of the accuracy of the measurements, the volume of the entire body was calculated by summing the volumes of the elliptical cylinders formed by the parallel transverse planes at the points of measurement. Based on the specific gravity of 1.06 the calculated weight of the body was 79.4 kg as compared to the actual weight of 79.5 kg.

Calculation of Resistance and SAR

The potential and body cross-section measurements were used to calculate the electrical conductivity and the resistance per unit length for various regions of the body. The resistance between two equal potential planes perpendicular to the axis of the body or body member is

$$R_{nm} = V_{nm}/I = \sum_{i=n}^{m} R(i)\,\Delta L \tag{9}$$

where R_{nm} is the resistance, V_{nm} is the measured voltage, I is the 10 mA current crossing the equipotential planes, ΔL is the 5 cm incremental distance between the measured cross-sections of the body, R(i) is the resistance per unit length for location i. R(i) is related to conductivity $\sigma(m)$ of a particular body section m by

$$R(i) = I/\sigma(m)A(i) \tag{10}$$

Thus the effective conductivity for the region between the equal potential planes may be obtained by equation (11)

$$\sigma = I/V_{nm} \sum_{i=n}^{m} \Delta L A(i) \tag{11}$$

In the above the combined cross sectional area of both lower limbs were used to give the linear resistance of the parallel combination of both lower limbs. The linear resistance for the arm corresponds to that for a single limb. The potential distribution V(k), the current density J(i) and the SAR(i) may be obtained for any current distribution by the equations (12) through (14) respectively

$$V(k) = \sum_{i=1}^{k} I(i)\,R(i) \tag{12}$$

$$J(i) = I(i)/A(i) \tag{13}$$

$$SAR(i) = J^2(i)/\sigma(m)\rho \tag{14}$$

where ρ is the density of the tissue assumed to be unity for the calculation of SAR values.

Computer algorithms were written to calculate the above parameters for the three types of current distributions measured by Deno (41) given in Figure 2 and equations 5 through 7. Calculations were also made for the case of a 1 milliampere current flowing through one arm, the body, and the feet in contact with the ground to simulate worst case conditions when the exposed person contacts an insulated object.

Calculations were made and tabulated for various exposure conditions corresponding to the current distributions in Figure 2. A sample of the results are tabulated in Table 8 covering cases where the subject is exposed with his feet in contact with the ground plane, free space, hands in contact with the ground while the feet are insulated, and hand in contact with a large object, such as a vehicle while the feet are grounded. For the latter case, a 1 mA current was assumed to be flowing through the arm, thorax, and the legs of the subject to ground. The values of current, current density, and potential resulting from such a contact with an object or vehicle may be

obtained by multiplying the tabulated values based on 1 mA by the object to ground current given in Table 5. The SAR may be obtained by multiplying by the square root of the object to ground current. The results were tabulated so that the data for a particular position could be related directly to the position along the body axis of a sketch of the subject next to the data. Tabulations were broken into two parts corresponding to the major axis of the body and legs and to the axis of the arm.

Though the sample of results are given in Table 8 for 20 kHz, The value of current at other frequencies may be assumed to vary approximately as frequency and the value of SAR as the square of frequency. The power absorption, P, in the exposed subject was determined for various body members and parts as well as for the whole body by simply summing the SAR values as shown in Equation 15.

$$P = \sum_i SAR(i)\,\Delta LA(i) \tag{15}$$

The average SAR was obtained by summing the absorbed powers in all body parts and dividing by body weight. The results are plotted as shown in Table 9 for various exposure situations. In order to compare the results for an ellipsoidal model of man as given in the Radio Frequency Radiation Dosimetry Handbook (20) the average measured apparent conductivity σ_a and loss factor, $e_2 = \sigma_a/2\pi f\epsilon\sigma_a$ was calculated and found to agree closely with that used in the Radio Frequency Radiation Dosimetry Handbook. The calculated average SAR, however, obtained from the VLF analysis was nearly a factor of 2 greater than that reported in the Radio Frequency Radiation Dosimetry Handbook The results, however, are consistent with experimental results of exposing scaled, realistic, human models to simulated VLF frequencies.

In addition to the calculations for various types of exposures, power absorption distributions in man exposed to VLF-MF fields while in contact with various vehicles was calculated based on short circuit currents given in Table 5. The results are tabulated in Table 10. In addition, the data corresponding to the 1 mA body current for a person in contact with an object given in Table 8 can be used to analyse the hazards associated with an exposed subject coming in contact with other types of objects, such as guy wires, rain gutters, construction equipment, cranes, and large animals. Methods for calculating the effective surface area of such objects can readily be found in the literature. For example, see Transmission Line Reference Book 345 kV and Above (18) and Deno (41).

For a given effective surface area, Equation 2 may be used to calculate the short circuit current and from this information one may obtain the absorbed powers, the distribution of SAR and the average SAR by simply multiplying the values in the Tables by the square of the short-circuit current in mA. As an example, we may consider vertical wires with an effective area given by

$$S = \pi \ell^2/\ln\left[(\ell/r)\left((4h+\ell)/(4h+3\ell)\right)^{1/2}\right] \tag{16}$$

and horizontal wires with an effective area given by

$$S = 2\pi h\,(\ell+2R)/\ln\,(2h/r) \tag{17}$$

ℓ is the length of the wire, r is the radius of the wire, and h is the distance of the wire from the surface of the ground. Since the worst case is obtained with vertical wires, we only consider that case as an example (results shown in Table 11). For this case, we considered a no. 9 gauge wire at a height of 1 meter above the ground with lengths varying from 1 to 30 meters.

DISCUSSION OF RESULTS AND RECOMMENDATIONS FOR SAFETY STANDARDS

The conductivity tabulated in Table 11 calculated from the distributed voltage measurements is highest in the limbs. This follows from the well known anisotropic characteristic of muscle fibers where the conductivity in the direction parallel to the fibers is greater than that perpendicular to the fibers. Conductivity appears relatively low near the neck and upper chest, which probably results from both the lower conductivity of brain and lung tissue and the presence of transverse currents resulting from the abrupt cross-sectional changes. Because of its large cross-section the resistance per unit length is lowest in the torso. Based on the body potential measurements, the body resistance along the entire axis varies from 262 ohms at 60 Hz to approximately 180 ohms at 200 kHz (according to Figure 7). Note that the currents and the SAR levels at the low frequencies are relatively low but increase dramatically to rather large values in the upper band.

The maximum electric field strength below levels of 1000 V/m that would violate any of the conditions given in the lower half of Table 2 is plotted in Figure 6. These conditions are: 1) The maximum current through any body member contacting ground or an object should not exceed levels equivalent to those allowed by the National Electric Safety Code in the frequency range where shock hazards may occur. 2) Total possible current entering the body should not exceed 200 mA for prevention of RF burns, and 3) The 0.4 W/kg average and 8 W/kg maximum SAR recommended by the ANSI RFPG shall not be exceeded. Based on the data in Tables 7 and 8, none of the conditions would be violated for free field exposures at 1 kV/m electric field strength and above. For cases where the body is not in contact with the ground or an object, 200 mA or more current through the body may not necessarily be a hazard. It should be kept in mind,

however, that this analysis is based on a homogeneous model. In actual tissue the SAR and current density could be significantly higher due to the shunting of current around low water content tissue through high water content tissue. Generally RF burns occur when the current is passing through a small cross-section of the body such as the fingers. In order to provide the criterion for situations where the body is not in contact with the ground we recommend restricting the maximum current density to 6.7 mA/cm^2 occurring in the wrist for a 200 mA current passing through the arm. If such a criterion is used based on the data in Table 7, the current density in a subject exposed on a ground plane would exceed the approximately 6.7 mA/cm^2 level for exposure between 2 and 3 MHz. Thus the field strength would have to be reduced to approximately 670 V/m at 3 MHz to prevent the current density from exceeding 6.7 mA/cm^2. Below 2 MHz, however, the field strength could reach 1 kV/m without violating the criterion. Thus a safe exposure standard for this exposure condition would correspond to the top solid curve in Figure 6, labeled number 1. Based on the data in Table 8 the field strength would have to be reduced to 650 V/m at 3 MHz (dashed line) for this case to prevent the average SAR from exceeding the ANSI standard of 0.4 W/kg and based on Table 7 the field strength would have to be limited to 629 V/m (dash dot line) at 3 MHz to prevent the maximum SAR from exceeding 8 W/kg (in the ankles). It is interesting to note that the electric field strength restriction is nearly identical to satisfy all three of the bioeffects criteria and is nearly identical to the maximum exposure specification of the ANSI RFPG.

When a subject is exposed with his hand in contact with the ground, however, Tables 7 and 8 indicate that one would have to limit the electric field strength to lower values. The average SAR criterion would be satisfied by restricting the electric field as shown by the dashed line No. 2 in Figure 6 (448 V/m at 3 MHz) while the RF burn and maximum SAR criteria would be satisfied by the respective No. 2 solid (222 V/m at 3 MHz) and the dash dot (238 V/m at 3 MHz) lines in Figure 6. Based on Table 4, the RF burn hazard and excessive SAR levels for persons coming in contact with vehicles under electric field exposure of significantly less than 1 kV/m strength becomes pronounced.

Line groups 3, 4 and 5 of Figure 6 indicate restrictions that would have to be placed on field strength to protect a perfectly grounded person coming in contact with automobiles, buses, and large trucks (trailer types). The fields would have to be limited by the solid lines for prevention of RF burns, by the dashed lines to satisfy the ANSI average SAR criterion, and by the dash dot lines to satisfy the ANSI maximum SAR 8 W/kg criterion. In addition to the RF burn and excessive SAR hazard, there is an electric shock hazard at frequencies below 100 to 200 kHz. The solid curves with cross marks at the left side of Figure 8 illustrates the restrictions on electric field strength for preventing the perception of electric shock when grounded exposed persons contact vehicles of various size based on the National Electric Code for fixed appliances at 60 Hz as given in Table 3. The short dashed lines specify the maximum fields to prevent a painful shock (Table 2) from occurring when exposed subjects contact vehicles. This is based on extrapolation of the National Electrical Code for limiting current from vehicles located under 60 Hz power transmission lines (Table 4). No direct criterion exist for predicting the change in let-go currents with frequency above 10 kHz (Figure 2). Thus, in the calculation of the specification for let-go currents we assumed approximately the same change with frequency as that for the threshold for perception.

For occupational exposures or limited infrequent general population exposures, restrictions according to the short dashed lines should be adequate for protection against shock. For continuous or frequent general population exposures restrictions according to the solid curves would be preferable for prevention of any sensation from shock. Even greater restrictions would be required for prevention of injury to exposed subjects in contact with wires as can be seen from Table 7. Generally, however, one must be in very close proximity to VLF-MF sources to experience the level of currents and SAR that violates the safety criteria.

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TABLE 1

WESTERN COUNTRY EXPOSURE STANDARDS .01 TO 3.0 MHz

	NATO	NATO	ANSI	ACGIH
FREQ. (MHz)	.01 - 1.0	1.0 - 3.0	.3 - 3.0	.01 - 3.0
E FIELD (V/m)	1000	500	614	614
H FIELD (A/m)	2.65	1.3	1.63	1.63
PWR. DEN. (mW/cm^2)	265	66	100	100

	USAF	NIOSH	MASS.	PORTLAND
FREQ. (MHz)	.01 -3.0	.3 - 3.0	.3 - 3.0	.1 - 3.0
E FIELD (V/m)	434	307 - 205	275	43
H FIELD (A/m)	1.15	.814-.544	.729	.114
PWR. DEN. (mW/cm^2)	50	25 - 11	20	.5

EXPOSURE STANDARDS 0.03 TO 3 MHz (USSR & EASTERN)

	POLAND	CZECH	USSR(OCC)	USSR(GP)
FREQ (MHz)	.1 - 3.0	.03 - 3.	.06 - 1.5 1.5 - 3.	.03 - .3 .3 - 3.
E FIELD (V/m)	20 -1000	50	50 50	20 10
H FIELD (A/m)	2 - 250		5	
PWR. DEN.	.1 - 265 (E)	.66 (E)	.66 (E)	.1 (E)
(mW/cm^2)	151 - 2.4×10^6(H)		943 (H)	.027 (H)

TABLE 2. SUMMARY OF ELECTRIC CURRENT EFFECTS ON HUMANS

ELECTRIC SHOCK		.06 kHz CURRENT mA.	10. kHz CURRENT mA.	SPARK DISCHARGE ENERGY
NO SENSATION IN HAND	MEN	.4	7.	
	WOMEN	.3	5.	
THRESHOLD OF PERCEPTION	MEN	1.1	12.	.12 mJ C=1000 pF V=350V
	WOMEN	.7	8.	
SHOCK--NOT PAINFUL	MEN	1.8	17.	.5-1.5 mJ C=1000 pF V=.7-1.2kV
	WOMEN	1.2	11.	
PAINFUL SHOCK MUSCLE CONTROL	MEN	9.	55.	
	WOMEN	6.	37.	
	CHILDREN	5.	27.	
PAINFUL SHOCK LET-GO THRESHOLD	MEN	16.	75.	
	WOMEN	10.5	50.	
SEVERE SHOCK	MEN	23.	94.	
	WOMEN	15.	63.	
THRESHOLD FOR RADIO FREQUENCY BURNS			200 mA.	

TABLE 3. MAXIMUM 60 Hz CURRENTS ALLOWED TO HUMAN BODY BY NATIONAL ELECTRICAL CODE (mA.) AND EQUIVALENT LEVELS AT OTHER FREQUENCIES

SOURCE	MAX 60Hz	EQUIVALENT 10kHz	EQUIVALENT 50kHz	EQUIVALENT 100kHz
PORTABLE APPLIANCES	.5	5.	20.	40.
FIXED APPLIANCES	.75	8.0	32.	64.
VEHICLES UNDER TRANSMISSION LINES	5.	27.	-	-
ANSI C95.1-1982 STANDARD	MAX SAR=	0.4W/kg avg	8.0W/kg peak	

TABLE 4. Summary of Bioeffects of VLF-MF EM Fields (Western literature)

Reference	Subject	Frequency	Intensity	Modulation	Exposure	Effects
Takashima (1966)	alcohol dehydrogenase	1-60 MHz	(200V)	50 us to 5 ms pulse	1 hr.	no effect on enzyme activity
	calf thymus DNA	10 Hz-10 kHz 100 kHz-10 MHz	(300V)	50 us to 5 ms pulse	1 hr	no effect on DNA structure
Geddes et al. (1971)	Human	10 kHz	.34 mA/cm2 (neck-abdomen) 12.7 mA/cm2 (trans chest)	CW		Threshold of sensation
	dog	10 kHz	>4 mA	CW		Threshold of ventricular fibrillation
Wyss and Boeckmann (1970)	Frog nerve	5-82.5 kHz	relative intensity voltage 1-5 (transverse) 1-25 (longitudinal) current 1-8 (transverse) 1-35(longitudinal)	3 us pulse		Threshold of excitation
	Frog muscle	5-82.5 kHz	voltage 1-10(transverse) 1-20 (longitudinal) current 1-20(transverse) 1-25 (longitudinal)	10 ms pulse		Threshold of excitation
Straub et al. (1972)	sea animals	10 Hz-25 kHz	2.5-60 mA/cm2	CW		Threshold of gross movement
Zervins (1973)	chick embryos	26 kHz	160 Gauss	CW	1 hr/day, 19 days	No effect on hatchability and development
Bollinger et al. (1974)	mice	25 kHz	15 kV/m, 7.5 A/m 10.6 kV/m, 5.3A/m	CW	1 hr/day 5 dys/wk, 50 days	No effect on reproduction growth, and metabolism
					1 hr/day 10,20,80, 70, 100 hrs	No effect on hematological parameters, organ weight, histology, chromosome and mitosis. The only possible effect was on immune system
Friend et al. (1975)	amoeba	100 kHz	200 V/m	CW	10 min	Change in shape, orientation and mobility
Gotz and Gotz (1977)	drosophila	9.6 kHz	2.5 gauss	CW or AM	54 days	No effect on mutation or development
Takashima et al. (1979)	Rabbit	1-10 MHz	0.5-1 kV/m	15 Hz AM	2 hr/day 6 weeks	Effect on EEG
		1-3 MHz	0.5-1 kV/m	15 AM	2-3 hrs.	No effect on EEG
Takashima Meleis (1981)	Rat	1, 3.5, 10, 35, 65, 100 MHz	200-250 V/m	14 Hz AM	3 hr/day	1 MHz fields affects EEG
Bergeron (1981)	Human blood	25 kHz	1160 A/m	CW	10 hrs	No effect on growth and

TABLE 5. SHORT CIRCUIT CURRENTS FOR OBJECTS EXPOSED TO VLF - MF f(kHz) 1 kV/m FIELDS

OBJECT	I(mA)	OBJECT	I(mA)
TRACTOR	1f	10m VERTICAL WIRE	2.08f
CAR	1.47f	20m VERTICAL WIRE	7.72f
JEEP	1.8f	10m CRANE	7.1f
BUS	6.5f	30m CRANE	44f
TRUCK	9.6f	100m HORIZ. WIRE	3.8f
5m VERTICAL WIRE	.561f	MAN	.26f

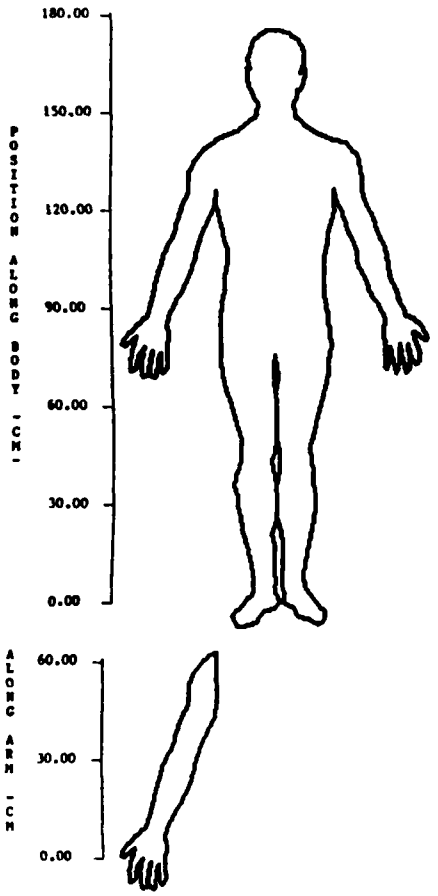
TABLE 6. COMPARISON OF MEASURED AND THEORETICAL SHORT CIRCUIT BODY CURRENT FOR MAN EXPOSED TO VLF-MF ELECTRIC FIELDS WITH FEET GROUNDED

LOCATION AND E-FIELD STRENGTH	FREQUENCY (kHz)	MEASURED CURRENT (mA/(kV/m))	THEORETICAL CURRENT (mA/(kV/m))
WET ASPHALT			
HAIKU, HAWAII OMEGA STATION, E=1 kV/m, HEIGHT OF SUBJECT=1.77m	10.2	2.9	2.88
LUALUALEI, HAWAII NAVAL VLF STATION, E=1 TO 2 kV/m HEIGHT OF SUBJECT=1.77m	23.4	6.75	6.60
JIM CREEK, WASHINGTON NAVAL VLF STATION E = 487 V/m			
SUBJECT #1 (HEIGHT=1.77m) (WEIGHT 80 kg)	24.8	6.0	6.60
SUBJECT #2 (HEIGHT=1.77m) (WEIGHT=80 kg)	24.8	5.6	6.60
SUBJECT #3 (HEIGHT=1.68m) (WEIGHT=64 kg) CORRECTED FOR 1.77m HEIGHT	24.8	6.22	6.60
RIGHT HAND OVER HEAD			
SUBJECT #1	24.8	7.23	7.39
SUBJECT #2	24.8	6.27	7.39
SUBJECT #3	24.8	6.97	7.39
LUALUALEI, HAWAII NAVAL LF STATION, E=76 V/m HEIGHT OF SUBJECT=1.77m	146.08	35.5	41.2
BROADCAST STATIONS LAS VEGAS NEV. BY TELL ET. AL., SUBJECT HEIGHT = 1.68m, CURRENT CORRECTED FOR HEIGHT OF 1.77m E=0.1 TO 63. V/m	720.	216.	203.
	920.	316.	259.
	1145.	366.	322.
	1350.	405.	380.
	1470.	560.	414.

TABLE 7. COMPARISON OF MEASURED AND THEORETICAL PERSON TO VEHICLE CURRENT RESULTING FROM VLF-MF ELECTRIC FIELD EXPOSURE

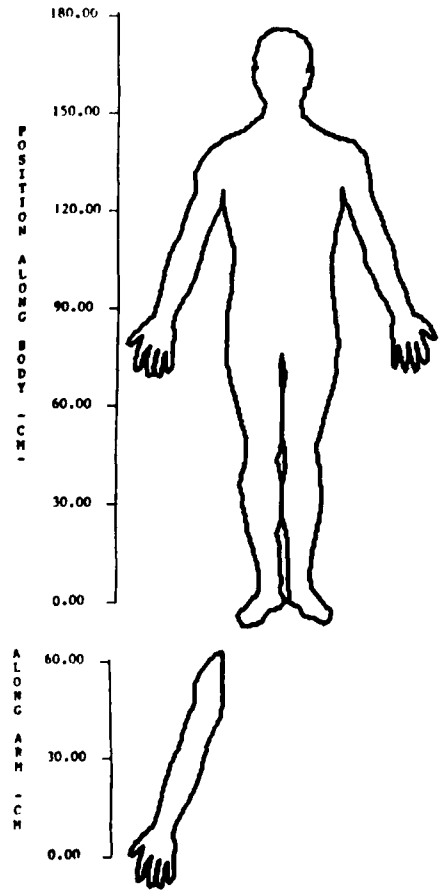
TYPE OF VEHICLE AND EXPOSURE CONDITIONS	FREQUENCY (kHz)	MEASURED CURRENT (mA/(kV/m))	THEORETICAL S.C. CURRENT (mA/(kV/m))
STANZA S.C. CURRENT WET ASPHALT, E= .92 kV/m OPEN CIRCUIT VOLTAGE MEAS.=240 THEORY=307 V	10.2	12.7	15.
BARE FEET	10.2	11.5	
WET GROUND S.C. CURRENT E=1.kV/m	23.4	28.	32.5
BARE FEET	23.4	29.7	
IMPALA S.C. CURRENT TO GROUND STRIP, E = .487 kV/m OPEN CIRCUIT VOLTAGE MEAS. 230 V	24.8	28.	
VOLKSWAGEN VAN S.C. CURRENT, WET ASPHALT E=.92 kV/m	10.2	18.8	18.4
BARE FEET	10.2	19.0	
LEATHER SHOES	10.2	12.6	
DRY ASPHALT, LEATHER SHOES	10.2	4.9	
RUBBER BEACH SANDELS WET ASPHALT	10.2	2.1	
CHEVROLET SPORTS VAN S.C.CURRENT, E=.92 kV/m	10.2	18.5	18.4
BARE FEET	10.2	18.4	
DRY ASPHALT, LEATHER SHOES	10.2	4.0	
BEACH SANDELS	10.2	0.8	
DODGE TRADEMAN 300 VAN E=1 kV/m, S.C. CURRENT, OPEN CIRC. V=415, WET GROUND	23.4	65.	42.5
BARE FEET	23.4	65.	
LEATHER SHOES	23.4	25.	

TABLE 8 EXPOSURE OF MAN TO 20.000 kHz 1kV/m ELECTRIC FIELDS WITH FEET GROUNDED



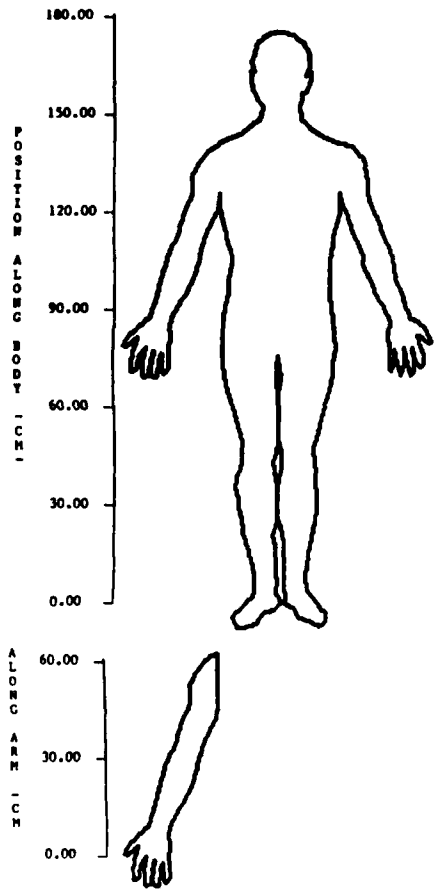
POSITION (cm)	CURRENT (mA)	CONDUCTIVITY S/m	RESISTANCE OHMS/cm	VOLTAGE V	CURRENT mA/cm2	SAR W/kg
175.	0.00E-01	1.77E-01	5.65E+00	9.10E-01	0.00E-01	0.00E-01
170.	8.51E-01	1.77E-01	2.91E+00	8.98E-01	4.38E-03	1.02E-05
165.	1.15E+00	1.77E-01	2.27E+00	8.85E-01	4.64E-03	1.15E-05
160.	1.36E+00	2.47E-01	1.92E+00	8.72E-01	6.44E-03	1.58E-05
155.	1.56E+00	2.47E-01	3.08E+00	8.47E-01	1.19E-02	5.41E-05
150.	1.69E+00	2.47E-01	1.20E+00	8.37E-01	5.01E-03	9.60E-06
145.	2.41E+00	2.10E-01	5.40E-01	8.31E-01	2.73E-03	3.35E-06
140.	3.28E+00	2.10E-01	1.39E+00	8.08E-01	9.61E-03	4.15E-05
135.	3.62E+00	2.77E-01	7.24E-01	7.95E-01	7.26E-03	1.79E-05
130.	3.86E+00	2.77E-01	4.88E-01	7.85E-01	5.22E-03	9.29E-06
125.	4.04E+00	2.77E-01	4.94E-01	7.75E-01	5.53E-03	1.04E-05
120.	4.24E+00	2.77E-01	5.24E-01	7.64E-01	6.16E-03	1.29E-05
115.	4.36E+00	2.77E-01	5.68E-01	7.52E-01	6.87E-03	1.61E-05
110.	4.44E+00	2.77E-01	5.50E-01	7.40E-01	6.76E-03	1.56E-05
105.	4.56E+00	4.55E-01	3.26E-01	7.32E-01	6.78E-03	9.52E-06
100.	4.65E+00	4.55E-01	3.15E-01	7.25E-01	6.66E-03	9.19E-06
95.	4.75E+00	4.55E-01	3.10E-01	7.18E-01	6.70E-03	9.31E-06
90.	4.82E+00	4.55E-01	3.90E-01	7.08E-01	8.54E-03	1.51E-05
85.	4.87E+00	3.55E-01	5.43E-01	6.95E-01	9.39E-03	2.34E-05
80.	4.93E+00	3.55E-01	5.55E-01	6.81E-01	9.71E-03	2.51E-05
75.	4.99E+00	3.55E-01	6.07E-01	6.66E-01	1.08E-02	3.08E-05
70.	5.05E+00	3.55E-01	6.73E-01	6.49E-01	1.21E-02	3.86E-05
65.	5.10E+00	3.55E-01	8.57E-01	6.27E-01	1.55E-02	6.40E-05
60.	5.16E+00	3.55E-01	1.05E+00	6.00E-01	1.92E-02	9.82E-05
55.	5.22E+00	3.47E-01	1.25E+00	5.68E-01	2.26E-02	1.39E-04
50.	5.26E+00	3.47E-01	1.43E+00	5.30E-01	2.61E-02	1.86E-04
45.	5.31E+00	3.47E-01	1.50E+00	4.90E-01	2.76E-02	2.07E-04
40.	5.34E+00	3.47E-01	1.27E+00	4.56E-01	2.35E-02	1.51E-04
35.	5.40E+00	3.47E-01	1.18E+00	4.24E-01	2.21E-02	1.32E-04
30.	5.43E+00	3.47E-01	1.53E+00	3.83E-01	2.87E-02	2.24E-04
25.	5.45E+00	3.47E-01	2.10E+00	3.26E-01	3.97E-02	4.27E-04
20.	5.48E+00	3.47E-01	2.64E+00	2.53E-01	5.03E-02	6.87E-04
15.	5.52E+00	3.47E-01	3.60E+00	1.54E-01	6.89E-02	1.29E-03
10.	5.54E+00	3.47E-01	3.45E+00	5.86E-02	6.63E-02	1.20E-03
5.	5.57E+00	3.47E-01	1.26E+00	2.34E-02	2.44E-02	1.62E-04
0.	5.60E+00	3.47E-01	8.35E-01	0.00E-01	1.62E-02	7.16E-05
55.	7.84E-01	4.07E-01	2.71E+00	7.95E-01	8.66E-03	1.74E-05
50.	7.78E-01	4.07E-01	3.33E+00	7.82E-01	1.05E-02	2.57E-05
45.	7.50E-01	4.07E-01	3.56E+00	7.69E-01	1.09E-02	2.74E-05
40.	7.11E-01	4.07E-01	4.01E+00	7.54E-01	1.16E-02	3.13E-05
35.	6.61E-01	4.07E-01	4.56E+00	7.39E-01	1.23E-02	3.48E-05
30.	5.94E-01	4.07E-01	4.34E+00	7.26E-01	1.05E-02	2.54E-05
25.	5.15E-01	4.19E-01	4.17E+00	7.16E-01	9.00E-03	1.82E-05
20.	4.26E-01	4.19E-01	5.06E+00	7.05E-01	9.02E-03	1.83E-05
15.	3.25E-01	4.19E-01	6.01E+00	6.95E-01	8.18E-03	1.51E-05
10.	2.18E-01	4.19E-01	7.99E+00	6.86E-01	7.31E-03	1.20E-05
5.	1.12E-01	4.19E-01	7.96E+00	6.82E-01	3.74E-03	3.14E-06
0.	0.00E-01	4.19E-01	1.17E+00	6.82E-01	0.00E-01	0.00E-01

TABLE 8 (CONTINUED) EXPOSURE OF MAN TO 20.000 kHz 1kV/m ELECTRIC FIELDS IN FREE SPACE



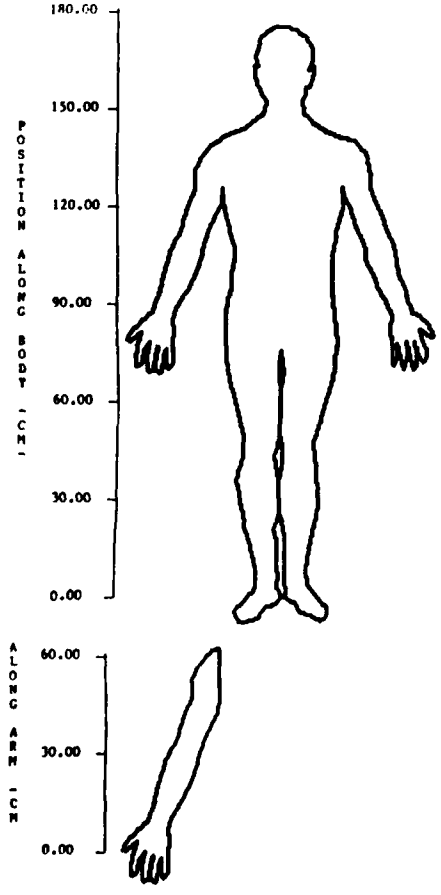
POSITION (cm)	CURRENT (mA)	CONDUCTIVITY S/m	RESISTANCE OHMS/cm	VOLTAGE V	CURRENT mA/cm2	SAR W/kg
175.	0.00E-01	1.77E-01	5.65E+00	3.77E-01	0.00E-01	0.00E-01
170.	5.53E-01	1.77E-01	2.91E+00	3.73E-01	2.85E-03	4.32E-06
165.	8.29E-01	1.77E-01	2.27E+00	3.66E-01	3.34E-03	5.94E-06
160.	9.60E-01	2.47E-01	1.92E+00	3.57E-01	4.54E-03	7.88E-06
155.	1.06E+00	2.47E-01	3.08E+00	3.41E-01	8.05E-03	2.48E-05
150.	1.12E+00	2.47E-01	1.20E+00	3.35F-01	3.34E-03	4.25E-06
145.	1.24E+00	2.10E-01	5.40E-01	3.32E-01	1.40E-03	8.84E-07
140.	1.42E+00	2.10E-01	1.39E+00	3.22E-01	4.17E-03	7.80E-06
135.	1.67E+00	2.77E-01	7.24E-01	3.17E-01	3.34E-03	3.81E-06
130.	1.81E+00	2.77E-01	4.88E-01	3.13E-01	2.45E-03	2.04E-06
125.	1.98E+00	2.77E-01	4.94E-01	3.08E-01	2.71E-03	2.51E-06
120.	2.11E+00	2.77E-01	5.24E-01	3.02E-01	3.07E-03	3.20E-06
115.	2.22E+00	2.77E-01	5.68E-01	2.96E-01	3.50E-03	4.17E-06
110.	2.32E+00	2.77E-01	5.50E-01	2.90E-01	3.53E-03	4.25E-06
105.	2.42E+00	4.55E-01	3.26E-01	2.86E-01	3.59E-03	2.67E-06
100.	2.55E+00	4.55E-01	3.15E-01	2.82E-01	3.65E-03	2.75E-06
95.	2.54E+00	4.55E-01	3.10E-01	2.78E-01	3.58E-03	2.66E-06
90.	2.56E+00	4.55E-01	3.90E-01	2.73E-01	4.54E-03	4.28E-06
85.	2.56E+00	3.55E-01	5.43E-01	2.66E-01	4.93E-03	6.47E-06
80.	2.56E+00	3.55E-01	5.55E-01	2.59E-01	5.04E-03	6.76E-06
75.	2.51E+00	3.55E-01	6.07E-01	2.52E-01	5.42E-03	7.79E-06
70.	2.48E+00	3.55E-01	6.73E-01	2.43E-01	5.91E-03	9.29E-06
65.	2.44E+00	3.55E-01	8.57E-01	2.33E-01	7.43E-03	1.47E-05
60.	2.41E+00	3.55E-01	1.05E+00	2.20E-01	8.98E-03	2.14E-05
55.	2.37E+00	3.47E-01	1.25E+00	2.05E-01	1.02E-02	2.85E-05
50.	2.32E+00	3.47E-01	1.43E+00	1.88E-01	1.15E-02	3.60E-05
45.	2.28E+00	3.47E-01	1.50E+00	1.71E-01	1.19E-02	3.84E-05
40.	2.23E+00	3.47E-01	1.27E+00	1.57E-01	9.81E-03	2.62E-05
35.	2.16E+00	3.47E-01	1.18E+00	1.44E-01	8.83E-03	2.12E-05
30.	2.10E+00	3.47E-01	1.53E+00	1.28E-01	1.11E-02	3.35E-05
25.	2.03E+00	3.47E-01	2.10E+00	1.06E-01	1.48E-02	5.94E-05
20.	1.97E+00	3.47E-01	2.64E+00	7.96E-02	1.80E-02	8.86E-05
15.	1.90E+00	3.47E-01	3.60E+00	4.48E-02	2.38E-02	1.53E-04
10.	1.76E+00	3.47E-01	3.45E+00	1.32E-02	2.10E-02	1.20E-04
5.	1.46E+00	3.47E-01	1.26E+00	3.05E-03	6.41E-03	1.12E-05
0.	0.00E-01	3.47E-01	8.35E-01	0.00E-01	0.00E-01	0.00E-01
55.	3.58E-01	4.07E-01	2.71E+00	3.17E-01	3.96E-03	3.63E-06
50.	3.56E-01	4.07E-01	3.33E+00	3.11E-01	4.82E-03	5.38E-06
45.	3.43E-01	4.07E-01	3.56E+00	3.05E-01	4.97E-03	5.73E-06
40.	3.25F-01	4.07E-01	4.01E+00	2.98E-01	5.31E-03	6.54E-06
35.	3.02F-01	4.07E-01	4.56E+00	2.91E-01	5.60E-03	7.28E-06
30.	2.71E-01	4.07E-01	4.34E+00	2.85E-01	4.79E-03	5.32E-06
25.	2.36F-01	4.19E-01	4.17E+00	2.79E-01	4.11E-03	3.81E-06
20.	1.95E-01	4.19E-01	5.06E+00	2.74E-01	4.13E-03	3.83E-06
15.	1.48E-01	4.19E-01	6.01E+00	2.69E-01	3.74E-03	3.15E-06
10.	9.98E-02	4.19E-01	7.99E+00	2.64F-01	3.34E-03	2.52E-06
5.	5.12F-02	4.19E-01	7.96E+00	2.61F-01	1.71E-03	6.56E-07
0.	0.00E-01	4.19E-01	1.17E+00	2.61F-01	0.00E-01	0.00E-01

TABLE 8 (CONTINUED) EXPOSURE OF MAN TO 20.000 kHz 1kV/m ELECTRIC FIELDS WITH HAND GROUNDED



POSITION (cm)	CURRENT (mA)	CONDUCTIVITY S/m	RESISTANCE OHMS/cm	VOLTAGE V	CURRENT mA/cm2	SAR W/kg
175.	0.00E-01	1.77E-01	5.65E+00	1.67E+00	0.00E-01	0.00E-01
170.	9.72E-01	1.77E-01	2.91E+00	1.65E+00	5.00E-03	1.33E-05
165.	1.26E+00	1.77E-01	2.27E+00	1.64E+00	5.07E-03	1.37E-05
160.	1.47E+00	2.47E-01	1.92E+00	1.63E+00	6.95E-03	1.85E-05
155.	1.64E+00	2.47E-01	3.08E+00	1.60E+00	1.25E-02	5.99E-05
150.	1.77E+00	2.47E-01	1.20E+00	1.59E+00	5.26E-03	1.06E-05
145.	1.88E+00	2.10E-01	5.40E-01	1.58E+00	2.14E-03	2.05E-06
140.	1.97E+00	2.10E-01	1.39E+00	1.57E+00	5.76E-03	1.49E-05
135.	1.83E+00	2.77E-01	7.24E-01	1.56E+00	3.67E-03	4.59E-06
130.	7.98E-01	2.77E-01	4.88E-01	1.56E+00	1.08E-03	3.96E-07
125.	7.98E-01	2.77E-01	4.94E-01	1.56E+00	1.09E-03	4.06E-07
120.	7.98E-01	2.77E-01	5.24E-01	1.56E+00	1.16E-03	4.57E-07
115.	7.98E-01	2.77E-01	5.68E-01	1.56E+00	1.26E-03	5.38E-07
110.	7.98E-01	2.77E-01	5.50E-01	1.55E+00	1.22E-03	5.04E-07
105.	7.98E-01	4.55E-01	3.26E-01	1.55E+00	1.19E-03	2.91E-07
100.	7.62E-01	4.55E-01	3.15E-01	1.55E+00	1.09E-03	2.47E-07
95.	7.26E-01	4.55E-01	3.10E-01	1.55E+00	1.02E-03	2.17E-07
90.	7.02E-01	4.55E-01	3.90E-01	1.55E+00	1.25E-03	3.21E-07
85.	6.78E-01	3.55E-01	5.43E-01	1.55E+00	1.31E-03	4.54E-07
80.	6.18E-01	3.55E-01	5.55E-01	1.55E+00	1.22E-03	3.94E-07
75.	5.70E-01	3.55E-01	6.07E-01	1.54E+00	1.23E-03	4.01E-07
70.	4.98E-01	3.55E-01	6.73E-01	1.54E+00	1.19E-03	3.76E-07
65.	4.62E-01	3.55E-01	8.57E-01	1.54E+00	1.41E-03	5.25E-07
60.	4.26E-01	3.55E-01	1.05E+00	1.54E+00	1.59E-03	6.70E-07
55.	3.72E-01	3.47E-01	1.25E+00	1.54E+00	1.61E-03	7.06E-07
50.	3.12E-01	3.47E-01	1.43E+00	1.53E+00	1.55E-03	6.53E-07
45.	2.76E-01	3.47E-01	1.50E+00	1.53E+00	1.44E-03	5.61E-07
40.	2.40E-01	3.47E-01	1.27E+00	1.53E+00	1.06E-03	3.04E-07
35.	1.98E-01	3.47E-01	1.18E+00	1.53E+00	8.09E-04	1.78E-07
30.	1.62E-01	3.47E-01	1.53E+00	1.53E+00	8.58E-04	2.00E-07
25.	1.26E-01	3.47E-01	2.10E+00	1.53E+00	9.16E-04	2.28E-07
20.	1.02E-01	3.47E-01	2.64E+00	1.52E+00	9.35E-04	2.38E-07
15.	7.80E-02	3.47E-01	3.60E+00	1.52E+00	9.74E-04	2.58E-07
10.	6.60E-02	3.47E-01	3.45E+00	1.52E+00	7.90E-04	1.70E-07
5.	3.60E-02	3.47E-01	1.26E+00	1.52E+00	1.58E-04	6.78E-09
0.	0.00E-01	3.47E-01	8.35E-01	1.52E+00	0.00E-01	0.00E-01
55.	6.00E+00	4.07E-01	2.71E+00	1.56E+00	6.63E-02	1.02E-03
50.	6.00E+00	4.07E-01	3.33E+00	1.46E+00	8.12E-02	1.53E-03
45.	6.00E+00	4.07E-01	3.56E+00	1.36E+00	8.69E-02	1.75E-03
40.	6.00E+00	4.07E-01	4.01E+00	1.24E+00	9.80E-02	2.23E-03
35.	6.00E+00	4.07E-01	4.56E+00	1.10E+00	1.11E-01	2.87E-03
30.	6.00E+00	4.07E-01	4.34E+00	9.71E-01	1.06E-01	2.60E-03
25.	6.00E+00	4.19E-01	4.17E+00	8.46E-01	1.05E-01	2.47E-03
20.	6.00E+00	4.19E-01	5.06E+00	6.94E-01	1.27E-01	3.64E-03
15.	6.00E+00	4.19E-01	6.01E+00	5.14E-01	1.51E-01	5.14E-03
10.	6.00E+00	4.19E-01	7.99E+00	2.74E-01	2.01E-01	9.09E-03
5.	6.00E+00	4.19E-01	7.96E+00	3.52E-02	2.00E-01	9.01E-03
0.	6.00E+00	4.19E-01	1.17E+00	0.00E-01	2.95E-02	1.96E-04

TABLE 8 (CONTINUED) EXPOSURE OF MAN TO 20.000 kHz 1 mA ELECTRIC CURRENT IN CONTACT WITH OBJECT



POSITION (cm)	CURRENT (mA)	CONDUCTIVITY S/m	RESISTANCE OHMS/cm	VOLTAGE V	CURRENT mA/cm2	SAR W/kg
175.	0.00E-01	1.77E-01	5.65E+00	1.51E-01	0.00E-01	0.00E-01
170.	0.00E-01	1.77E-01	2.91E+00	1.51E-01	0.00E-01	0.00E-01
165.	0.00E-01	1.77E-01	2.27E+00	1.51E-01	0.00E-01	0.00E-01
160.	0.00E-01	2.47E-01	1.92E+00	1.51E-01	0.00E-01	0.00E-01
155.	0.00E-01	2.47E-01	3.08E+00	1.51E-01	0.00E-01	0.00E-01
150.	0.00E-01	2.47E-01	1.20E+00	1.51E-01	0.00E-01	0.00E-01
145.	0.00E-01	2.10E-01	5.40E-01	1.51E-01	0.00E-01	0.00E-01
140.	0.00E-01	2.10E-01	1.39E+00	1.51E-01	0.00E-01	0.00E-01
135.	0.00E-01	2.77E-01	7.24E-01	1.51E-01	0.00E-01	0.00E-01
130.	1.00E+00	2.77E-01	4.88E-01	1.49E-01	1.35E-03	6.22E-07
125.	1.00E+00	2.77E-01	4.94E-01	1.47E-01	1.37E-03	6.37E-07
120.	1.00E+00	2.77E-01	5.24E-01	1.44E-01	1.45E-03	7.17E-07
115.	1.00E+00	2.77E-01	5.68E-01	1.41E-01	1.57E-03	8.44E-07
110.	1.00E+00	2.77E-01	5.50E-01	1.38E-01	1.52E-03	7.91E-07
105.	1.00E+00	4.55E-01	3.26E-01	1.37E-01	1.49E-03	4.57E-07
100.	1.00E+00	4.55E-01	3.15E-01	1.35E-01	1.43E-03	4.25E-07
95.	1.00E+00	4.55E-01	3.10E-01	1.34E-01	1.41E-03	4.12E-07
90.	1.00E+00	4.55E-01	3.90E-01	1.32E-01	1.77E-03	6.52E-07
85.	1.00E+00	3.55E-01	5.43E-01	1.29E-01	1.93E-03	9.87E-07
80.	1.00E+00	3.55E-01	5.55E-01	1.26E-01	1.97E-03	1.03E-06
75.	1.00E+00	3.55E-01	6.07E-01	1.23E-01	2.16E-03	1.24E-06
70.	1.00E+00	3.55E-01	6.73E-01	1.20E-01	2.39E-03	1.52E-06
65.	1.00E+00	3.55E-01	8.57E-01	1.15E-01	3.04E-03	2.46E-06
60.	1.00E+00	3.55E-01	1.05E+00	1.10E-01	3.73E-03	3.69E-06
55.	1.00E+00	3.47E-01	1.25E+00	1.04E-01	4.33E-03	5.10E-06
50.	1.00E+00	3.47E-01	1.43E+00	9.68E-02	4.97E-03	6.71E-06
45.	1.00E+00	3.47E-01	1.50E+00	8.93E-02	5.20E-03	7.36E-06
40.	1.00E+00	3.47E-01	1.27E+00	8.29E-02	4.40E-03	5.27E-06
35.	1.00E+00	3.47E-01	1.18E+00	7.71E-02	4.09E-03	4.54E-06
30.	1.00E+00	3.47E-01	1.53E+00	6.94E-02	5.30E-03	7.62E-06
25.	1.00E+00	3.47E-01	2.10E+00	5.89E-02	7.27E-03	1.44E-05
20.	1.00E+00	3.47E-01	2.64E+00	4.57E-02	9.17E-03	2.29E-05
15.	1.00E+00	3.47E-01	3.60E+00	2.77E-02	1.25E-02	4.24E-05
10.	1.00E+00	3.47E-01	3.45E+00	1.05E-02	1.20E-02	3.89E-05
5.	1.00E+00	3.47E-01	1.26E+00	4.17E-03	4.39E-03	5.23E-06
0.	1.00E+00	3.47E-01	8.35E-01	0.00E-01	2.90E-03	2.28E-06
55.	1.00E+00	4.07E-01	2.71E+00	1.51E-01	1.10E-02	2.83E-05
50.	1.00E+00	4.07E-01	3.33E+00	1.68E-01	1.35E-02	4.25E-05
45.	1.00E+00	4.07E-01	3.56E+00	1.86E-01	1.45E-02	4.87E-05
40.	1.00E+00	4.07E-01	4.01E+00	2.06E-01	1.63E-02	6.18E-05
35.	1.00E+00	4.07E-01	4.56E+00	2.29E-01	1.86E-02	7.98E-05
30.	1.00E+00	4.07E-01	4.34E+00	2.50E-01	1.76E-02	7.22E-05
25.	1.00E+00	4.19E-01	4.17E+00	2.71E-01	1.75E-02	6.86E-05
20.	1.00E+00	4.19E-01	5.06E+00	2.97E-01	2.12E-02	1.01E-04
15.	1.00E+00	4.19E-01	6.01E+00	3.27E-01	2.52E-02	1.43E-04
10.	1.00E+00	4.19E-01	7.99E+00	3.67E-01	3.35E-02	2.52E-04
5.	1.00E+00	4.19E-01	7.96E+00	4.06E-01	3.39E-02	2.50E-04
0.	1.00E+00	4.19E-01	1.17E+00	4.12E-01	4.91E-03	5.43E-06

TABLE 9. DISTRIBUTION OF POWER ABSORPTION (WATTS) IN MAN EXPOSED TO VLF-MF FIELDS 1 kV/m EXPOSURE, E FIELD PARALLEL LONG AXIS, 1 mA. CURRENT ASSUMED FOR CONTACT WITH OBJECT

FREQUENCY= 20.000 kHz

	FEET GROUNDED	FREE SPACE	HAND GROUNDED	HAND CONTACT WITH OBJECT
LOWER LEG	3.27E-03	4.31E-04	3.42E-06	1.10E-04
UPPER LEG	7.14E-04	1.67E-04	6.86E-06	2.77E-05
LOWER TORSO	2.13E-04	6.03E-05	4.98E-06	9.42E-06
MID TORSO	3.14E-04	7.78E-05	2.15E-05	1.48E-05
UPPER TORSO	1.38E-04	2.83E-05	4.87E-05	0.00E-01
NECK	8.81E-05	3.78E-05	9.08E-05	0.00E-01
HEAD	4.34E-05	2.11E-05	5.25E-05	0.00E-01
LOWER ARM	4.67E-05	9.75E-06	6.61E-03	1.83E-04
UPPER ARM	1.12E-04	2.35E-05	4.05E-03	1.13E-04
WHOLE BODY	4.94E-03	8.56E-04	1.09E-02	4.58E-04
AVG SAR W/kg	6.22E-05	1.08E-05	1.37E-04	5.77E-06

TABLE 10. DISTRIBUTION OF POWER ABSORPTION (WATTS) IN MAN EXPOSED TO 1 kV/m VLF-MF FIELDS WHILE IN CONTACT WITH VEHICLE WITH FEET GROUNDED

FREQUENCY= 20.000 kHz

	CAR	JEEP WAGON	SCHOOL BUS	SEMI-TRUCK
LOWER LEG	9.52E-02	1.43E-01	1.86E+00	4.06E+00
UPPER LEG	2.39E-02	3.59E-02	4.68E-01	1.02E+00
LOWER TORSO	8.14E-03	1.22E-02	1.59E-01	3.47E-01
MID TORSO	1.28E-02	1.91E-02	2.49E-01	5.44E-01
UPPER TORSO	0.00E-01	0.00E-01	0.00E-01	0.00E-01
NECK	0.00E-01	0.00E-01	0.00E-01	0.00E-01
HEAD	0.00E-01	0.00E-01	0.00E-01	0.00E-01
LOWER ARM	1.59E-01	2.38E-01	3.10E+00	6.76E+00
UPPER ARM	9.73E-02	1.46E-01	1.90E+00	4.15E+00
WHOLE BODY	3.96E-01	5.94E-01	7.74E+00	1.69E+01
AVG SAR W/kg	4.99E-03	7.48E-03	9.75E-02	2.13E-01

TABLE 11. DISTRIBUTION OF POWER ABSORPTION (WATTS) IN MAN EXPOSED TO VLF-MF FIELDS WHILE IN CONTACT WITH VERTICAL 9 GAUGE WIRE OF LENGTH L WITH LOWER END 1 m ABOVE GROUND

FREQUENCY= 20.000 kHz

	L= 1.m	L= 2.m	L= 3.m	L= 4.m
LOWER LEG	3.31E-05	4.41E-04	2.02E-03	6.00E-03
UPPER LEG	8.31E-06	1.11E-04	5.07E-04	1.51E-03
LOWER TORSO	2.83E-06	3.77E-05	1.72E-04	5.13E-04
MID TORSO	4.43E-06	5.90E-05	2.70E-04	8.04E-04
UPPER TORSO	0.00E-01	0.00E-01	0.00E-01	0.00E-01
NECK	0.00E-01	0.00E-01	0.00E-01	0.00E-01
HEAD	0.00E-01	0.00E-01	0.00E-01	0.00E-01
LOWER ARM	5.51E-05	7.34E-04	3.36E-03	9.99E-03
UPPER ARM	3.38E-05	4.50E-04	2.06E-03	6.13E-03
WHOLE BODY	1.38E-04	1.83E-03	8.39E-03	2.49E-02
AVG SAR W/kg	1.73E-06	2.31E-05	1.06E-04	3.14E-04
	L= 5.m	L= 10.m	L= 20.m	L= 30.m
LOWER LEG	1.39E-02	1.91E-01	2.63E+00	1.23E+01
UPPER LEG	3.48E-03	4.79E-02	6.60E-01	3.09E+00
LOWER TORSO	1.19E-03	1.63E-02	2.24E-01	1.05E+00
MID TORSO	1.86E-03	2.55E-02	3.52E-01	1.65E+00
UPPER TORSO	0.00E-01	0.00E-01	0.00E-01	0.00E-01
NECK	0.00E-01	0.00E-01	0.00E-01	0.00E-01
HEAD	0.00E-01	0.00E-01	0.00E-01	0.00E-01
LOWER ARM	2.31E-02	3.18E-01	4.37E+00	2.05E+01
UPPER ARM	1.42E-02	1.95E-01	2.68E+00	1.26E+01
WHOLE BODY	5.77E-02	7.93E-01	1.09E+01	5.11E+01
AVG SAR W/kg	7.26E-04	9.98E-03	1.38E-01	6.44E-01

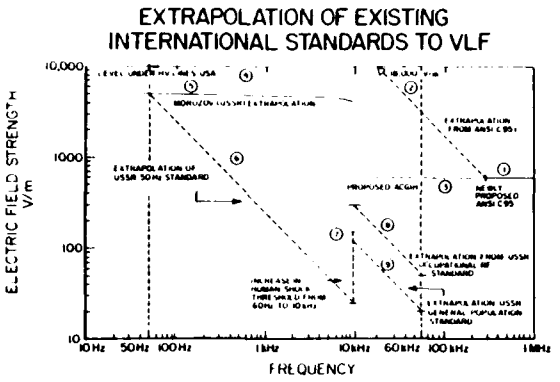


Fig. 1. Extrapolation of existing International Standards to VLF

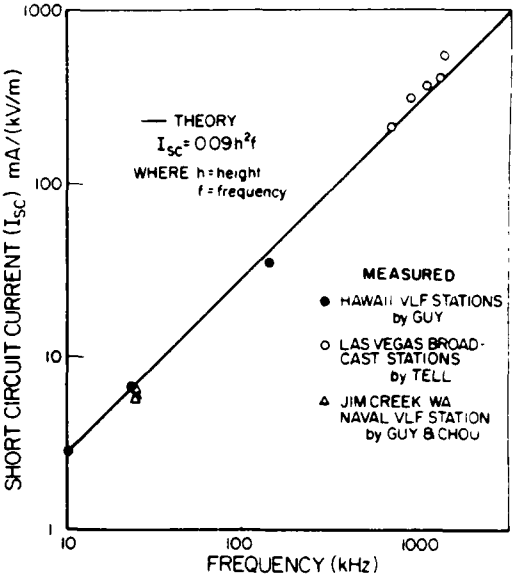


Fig. 4. Short circuit body current of grounded man exposed to VLF-MF electric fields

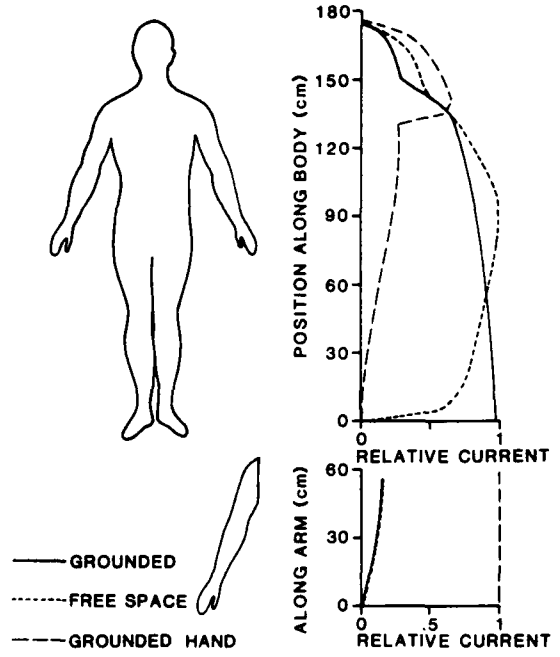


Fig. 2. Relative current distribution in man exposed to VLF-MF fields (41)

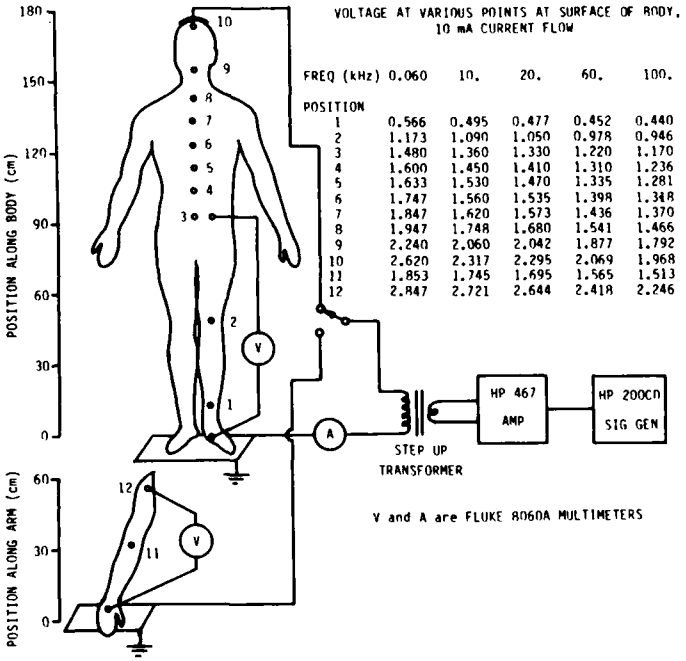


Fig. 5. Measurement of potential distribution

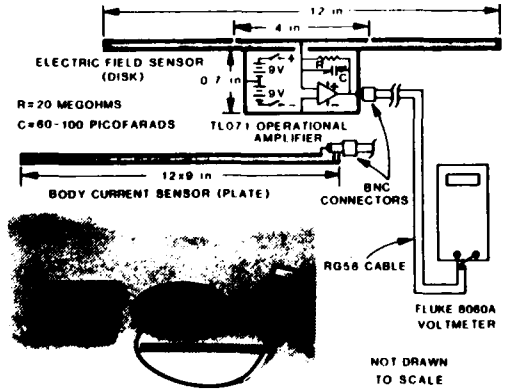


Fig. 3. Electric field and body current measuring equipment

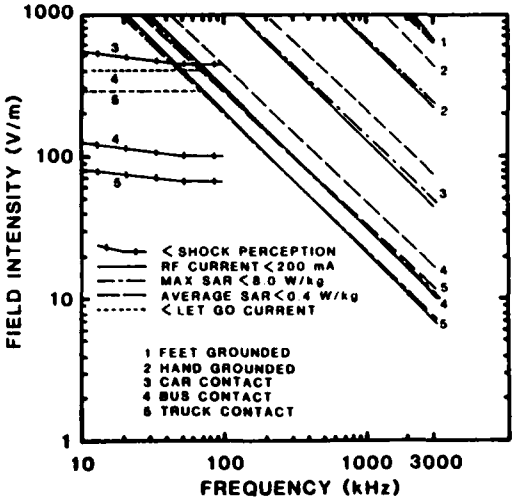


Fig. 6. Recommended maximum VLF-MF electric field exposure levels

DEVELOPMENT AND APPLICATION OF NEW RADIOFREQUENCY RADIATION SAFETY STANDARDS

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SUMMARY

Historically, an average incident power density of 100 mW/cm^2 of microwave radiation was considered hazardous and 10 mW/cm^2 was considered safe. Microwaves were loosely defined as electromagnetic emission at frequencies between 0.3 and 30 GHz. Research conducted over the past few years provides a better scientific basis for radiofrequency radiation (RFR) safety guidelines. RFR is defined to cover the frequency range from 10 kHz to 300 GHz and includes microwaves. Absorption and distribution of RFR are strongly dependent on the size of the irradiated object and the frequency of the incident energy. It has become common practice to report biological effects of RFR as a function of specific absorption rate (SAR) expressed as W/kg. The most widely held view is that the threshold for adverse effects lies above 4 W/kg. This value underlies the rationale for most standards that have emerged since 1982. Using a safety factor of 10, the American National Standards Institute developed RFR protection guides that limit all human whole-body exposures to incident energy that results in an SAR no greater than 0.4 W/kg. This allows incident average power densities from 1 to 100 mW/cm^2, depending on the frequency of the radiation. The American Conference of Governmental Industrial Hygienists applied the same safety factor to establish RF/microwave radiation threshold limit values for occupational workers. The Executive Council of the International Radiation Protection Association also approved interim guidelines to limit human exposures to RFR fields, using a whole-body SAR as the basic limits of exposure. Similar proposals for new RFR safety guidelines have been proposed by the National Radiological Protection Board of the United Kingdom. These new safety guidelines are compared with many other RFR standards used throughout the world today including NATO STANAG 2345. In general, the new RFR safety standards provide an added margin of safety over those previously used.

RADIOFREQUENCY RADIATION (RFR) SAFETY STANDARDS

INTRODUCTION

The development and application of devices that emit radiofrequency radiation (RFR) have significantly increased the quality of life throughout the world. Yet in recent years the beneficial aspects of radiofrequency/microwave technology have been somewhat overshadowed by a public arousal of the fear of potential adverse effects. This fear, in turn, has led to increased RFR research, resulting in a much better understanding of the interaction of RFR fields and biological systems, and has resulted in the promulgation of new RFR safety guidelines. The new exposure standards are based on what is known about the frequency-dependent nature of RFR energy deposition in biological systems and the current knowledge of biological effects. In general, the new safety guidelines provide an added margin of safety over what was previously used.

RFR is generally identified as nonionizing electromagnetic emission in the frequency range from 10 kHz to 300,000 MHz. Systems and devices that emit such radiation include a vast assortment of radar and communication systems, microwave ovens and other consumer devices, RF heat sealers, radio and television broadcast transmitters, and numerous medical devices for diagnostic and therapeutic purposes.

The inherent risks to health from RFR exposures are directly linked to the absorption and distribution of RFR energy in the body, and the absorption and distribution are strongly dependent on the size and orientation of the body and the frequency and polarization of the incident radiation (1). Both theory and experiment show that RFR absorption in prolate spheroid models approaches a maximum value when the long axis of the body is both parallel to the electric field vector and equal to approximately four-tenths of the wavelength of the incident RFR field (2). Thus, a "standard" 70-kg, 1.75-m human, exposed to a uniform plane-wave RFR field in free space with the E-field aligned with the long axis of the body, would absorb the most energy at a frequency of about 70 MHz (2). If the person were standing in contact with a conducting ground plane (producing a change in the apparent long-axis length), the frequency for maximum RFR absorption would be about 35 MHz. This frequency-dependent behavior is illustrated in Figure 1 for several human sizes (using prolate spheroid models having body masses of 10, 32, and 70 kg). The average whole-body specific absorption rate (SAR) in W/kg is plotted as a function of radiation frequency in MHz for an incident average power density of 1 mW/cm^2.

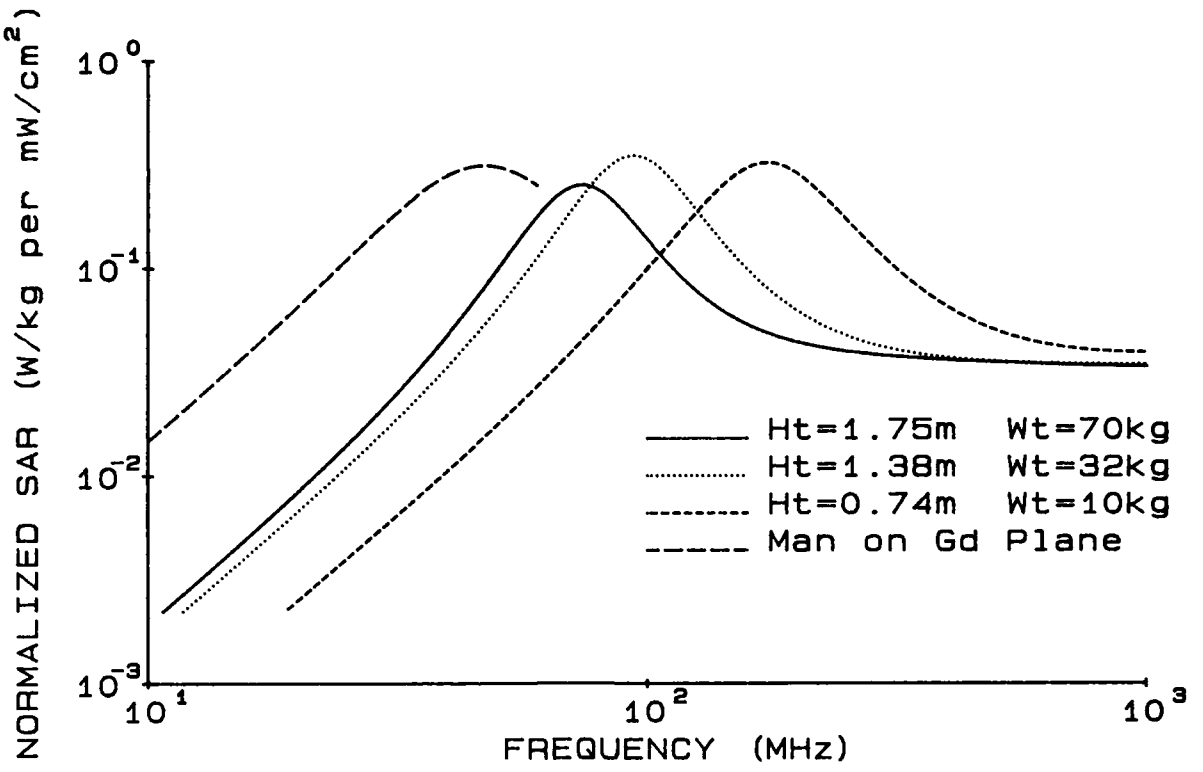

Figure 1. Specific absorption rate for different size humans

RFR SAFETY STANDARDS

United States RFR Safety Guidelines

American National Standards Institute (ANSI) Standard

For more than 20 years, the United States and most of the free world used a single field-intensity value to maintain the safety of personnel exposed to RFR. A power density of 10 mW/cm^2, time averaged over any 6-minute period, was applied as an acceptable exposure level; and it was generally thought to include a safety factor of 10. During the past 5-10 years, it has become well accepted that the absorption and distribution of RFR in humans are strongly dependent on the frequency of the incident radiation, as shown in Figure 1. Therefore, when ANSI revised its safety standard in 1982, it incorporated this frequency-dependency concept, using SAR as a common denominator for biological effects. The new ANSI standard, published 1 Sep 1982, covers the frequency range from 0.3 MHz to 100 GHz and allows average incident power densities from 1 to 100 mW/cm^2, depending on the radiation frequency. It limits the average whole-body absorption to 0.4 W/kg or less and the spatial peak SAR to 8 W/kg as averaged over any 1 g of tissue (3).

Figure 2 illustrates how the ANSI standard was derived. The relative power absorption curves illustrated in Figure 1 were used to establish the shape of the ANSI curve. It was normalized to 0.4 W/kg because the ANSI committee, after reviewing the biological effects data base, believed the threshold for adverse biological effects to be greater than 4 W/kg. Thus the 0.4 W/kg was selected to include a safety factor of 10. The ANSI RFR Protection Guides in terms of the mean squared electric (E^2) and magnetic (H^2) field strengths and in terms of the equivalent plane-wave free-space power density as a function of frequency are given in Table 1.

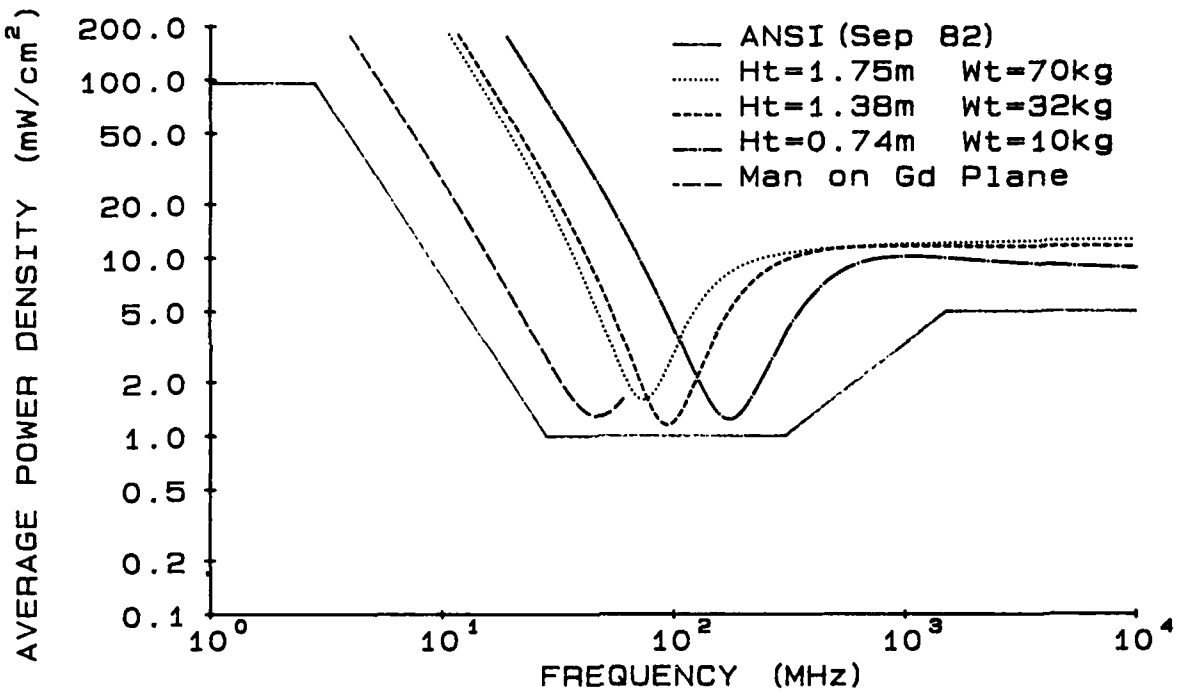

Figure 2. Power densities that limit human whole-body SAR to 0.4 W/kg compared to ANSI standard

TABLE 1
ANSI Radiofrequency Protection Guides

Frequency Range (MHz)	E^2 (V^2/m^2)	H^2 (A^2/m^2)	Power Density (mW/cm^2)
0.3 - 3	400000	2.5	100
3 - 30	$4000(900/f^2)$	$0.025(900/f^2)$	$900/f^2$
30 - 300	4000	0.025	1.0
300 - 1500	4000(f/300)	0.025(f/300)	f/300
1500 - 100000	20000	0.125	5.0

American Conference of Governmental Industrial Hygienists (ACGIH) TLV

In May 1983, ACGIH published new threshold limit values (TLVs) for radiofrequency /microwave radiation (4). Like the ANSI standards, the ACGIH TLVs limit human absorptions to an SAR of 0.4 W/kg or less, averaged over any 6-minute period. Unlike the ANSI standards, the TLVs cover the added frequency range from 10 to 300 kHz and from 100 to 300 GHz. Because the TLVs are to be applied in occupational settings, they assume that no children (small humans) will be in the workplace. This assumption allows an average incident power density of 10 mW/cm^2 at frequencies greater than 1 GHz, while maintaining the same 0.4-W/kg whole-body absorption limit. This can be seen from Figure 2 if the absorption curve for a 10-kg human is removed. The arbitrary 100-mW/cm^2 cap applied in the frequency range from 10 kHz to 3 MHz appears safe on the basis of whole-body SAR. However, RFR intensities of 100 mW/cm^2 may result in shocks and/or burns under certain conditions. The 100-mW/cm^2 limit should not restrict many operations and serves as a reminder that a person can begin to encounter potentially significant problems at such levels. The ACGIH TLV provides procedures to minimize these problems and to maintain personnel safety while reducing operational constraints. The ACGIH TLVs are established as safety guidelines for the workplace. They are intended for use in the practice of industrial hygiene and should be interpreted and applied only by a person trained in this discipline. The ACGIH TLVs are presented in Table 2.

TABLE 2
ACGIH radiofrequency/microwave threshold limit values

Frequency	Power Density (mW/cm^2)	E^2 (V^2/m^2)	H^2 (A^2/m^2)
10 KHz to 3 MHz	100	377,000	2.65
3 MHz to 30 MHz	$900/f^2$	$3770 \times 900/f^2$	$900/37.7\ f^2$
30 MHz to 100 MHz	1	3770	0.027
100 MHz to 1000 MHz	f/100	3770 x f/100	f/37.7 x 100
1 GHz to 300 GHz	10	37,700	0.265

mW/cm^2 = milliwatts per centimeter squared
f = frequency in MHz

Federal Guidelines

The United States has not established federal guidelines for RFR exposures at this time (Oct 1984). The voluntary guidelines offered by ANSI and ACGIH, coupled with those used by the individual services of the Department of Defense and some State standards, represent the RFR safety guidelines applied in the United States in the past few years (5,6).

International Radiation Protection Association Guidelines

On 8 July 1983, the Executive Council of the International Radiation Protection Association (IRPA) approved interim guidelines on limits of exposure to radiofrequency electromagnetic fields in the frequency range from 100 kHz to 300 GHz (7). The International Nonionizing Radiation Committee of IRPA included participants from France, Netherlands, Poland, Denmark, Germany, Great Britain, Australia, and the United States. Environmental Health Criteria 16, "Radiofrequency and Microwaves", published in 1981, serves as the primary scientific rationale for the development of the IRPA RFR guidelines (8). These guidelines apply to RFR exposure of occupational workers and the general public. The basic limits of exposure for frequencies greater than 10 MHz are expressed in whole-body averaged SAR. For practical purposes, derived limits of exposure expressed in average incident power density are also given. See Tables 3 and 4. The derived limits are extremely conservative in the frequency range 10-30 MHz. This approach, to state the exposure limit in terms of whole-body SAR, represents a departure from current practices; i.e., even though they are based on limiting the whole-body SAR, most new standards express the permissible exposure levels in average incident power density. For occupational workers, the IRPA exposure limit for frequencies greater than 10 MHz is 0.4 W/kg when averaged over any 6 minutes and over the whole body, or 4 W/kg when averaged over any 6 minutes in any 1 g of tissue. For the general public, the IRPA exposure limit is five times lower; i.e., 0.08 W/kg when averaged over any 6 minutes and over the whole body or 0.8 W/kg when averaged over any 6 minutes and any 1 g of tissue.

TABLE 3
IRPA occupational exposure limits to radiofrequency electromagnetic fields

Frequency MHz	E (V/m)	H (A/m)	Power Density (mW/cm^2)
0.1-1	194	0.51	*10
>1-10	$194/f^{1/2}$	$0.51/f^{1/2}$	*10/f
>10-400	61	0.16	1
>400-2000	$3\ f^{1/2}$	$0.008f^{1/2}$	f/400
>2000-300,000	137	0.36	5

* These values are provided for information only and are not to be considered for determining compliance.

TABLE 4
IRPA general public exposure limits to radiofrequency electromagnetic fields

Frequency MHz	E (V/m)	H (A/m)	Power Density mW/cm^2
0.1-1	87	0.23	*2
>1-10	$87/f^{1/2}$	$0.23/f^{1/2}$	*2/f
>10-400	27.5	0.073	0.2
>400-2000	$1.375f^{1/2}$	$0.0037f^{1/2}$	f/2000
>2000-300,000	61	0.16	1

*Same note as above

Figure 3 presents a comparison of the ANSI, ACGIH, and IRPA, RFR safety standards plotted as average incident power density versus frequency. These standards are all based on the assumption that 4 W/kg is a reasonable threshold for adverse biological effects. Differences in the permissible incident power densities as a function of frequency result from the degree of conservatism applied in each instance.

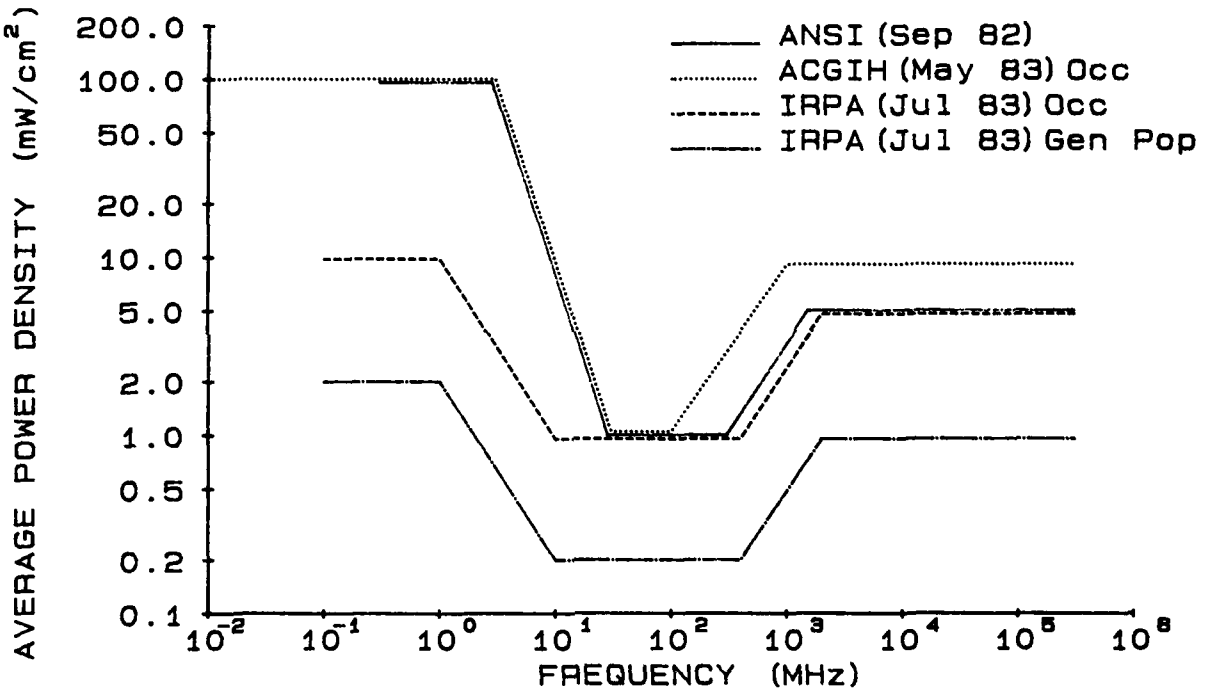

Figure 3. Comparison of RFR safety guidelines based on an adverse effect threshold of 4 W/kg

United Kingdom Safety Standards

For many years the United Kingdom (UK) used the 10-mW/cm^2 standard for RFR exposures over the frequency range from 30 MHz to 30 GHz. In 1983 the National Radiological Protection Board of the UK proposed new safety guidelines. The proposed limits for continuous exposure of adult populations are essentially the same as the ACGIH TLV standard previously described. The proposed limits for the general population, including children, are essentially the same as the ANSI standard, except the UK proposal extends the lower frequency limit down to 3 kHz and the upper frequency limit to 300 GHz. The proposals were consultative and comments were invited. These proposals and the rationale used are described in Radiological Protection Bulletin No. 52 dated May 1983 (9).

Canadian Standards

The Canadian federal government also used the so called 10-mW standard until 1979; then it revised its standard to include separate occupational and nonoccupational guidelines (10). The maximum permissible exposure level for the general public (nonoccupational) is 1 mW/cm^2, when averaged over a one-minute period, for

the frequency range 10 MHz to 300 GHz. Also the rms electric field strength must not exceed 60 V/m. For occupational situations, the maximum exposure levels are frequency and time dependent. From 10 MHz to 1 GHz, the Canadian RFR standard permits exposure to 1 mW/cm^2 for an 8-hour work day, 10 mW/cm^2 for 6 minutes or less, and 25 mW/cm^2 for 2.4 minutes or less. From 1 to 300 GHz, exposures of 5 mW/cm^2 for an 8-hour work day, 10 mW/cm^2 for 30 minutes, and 25 mW/cm^2 for 2.4 minutes are permitted.

USSR Standards

RFR exposure limits in the USSR and other Warsaw Pact countries are generally recognized as being lower than those of Western countries. This results primarily from differences in philosophy and processes for setting standards (11). In the United States, safety factors are usually applied to values at which biologically significant effects are observed in laboratory animal studies. In the USSR, the principle of "no effect," adverse or otherwise, on any person appears to be the primary basis for standard setting. A recent publication of the World Health Organization states, "The USSR occupational and public safety standards are based on the principle of complete prevention of health risks, and, therefore, include large safety factors" (8). Until recently, the maximum level for 24-hour exposure of the general population was 5 $\mu W/cm^2$. Recent visitors to the USSR report that this has been changed to 10 $\mu W/cm^2$. The occupational exposure levels are summarized in Table 5 (9). The Soviet military organizations are exempt from such standards, but it is likely that they use RFR exposure levels 10 times higher for their military guidelines. Other Warsaw Pact countries, such as the German Democratic Republic and Bulgaria, appear to use essentially the same RFR standards as the USSR.

TABLE 5
USSR Maximum Levels for Occupational Exposure to Radiofrequency Radiation

Frequency (GHz)	Exposure Duration	Exposure Limit
0.01 to 0.03	Working Day	20 V/m
0.03 to 0.05	Working Day	10 V/m 0.3 A/m
0.05 to 0.3	Working Day	5 V/m 0.15 A/m
0.3 to 300	Working Day	[a] 0.01 mW/cm^2 [b]
	Working Day	0.1 mW/cm^2 [c]
	2 h	0.1 mW/cm^2 [b]
	2 h	1 mW/cm^2 [c]
	20 min	1 mW/cm^2 [b]

[a]Using the recent proposals that the product of average power density and exposure time should not exceed 720 $mW\text{-}s/cm^2$, the exposure limit becomes 0.025 mW/cm^2 instead of 0.01 mW/cm^2.

[b]Stationary antennas

[c]Rotating antennas

In March 1983 B.M. Savin reported on proposed changes to the USSR microwave exposure standards (12). The proposals served as the basis for developing Amendment No. 1 to State Standard 12.1.006-76 CCBT "The Electromagnetic Fields of Radio Frequency--General Requirements." The average power density is still the basic parameter used to assess compromise of state-of-health, but maximum power density is to be determined by the allowable energy load on the body and the exposure duration. The maximum power density will be determined by dividing the energy density by the exposure time. The maximum energy density allowed is equal to 720 $mW\text{-}s/cm^2$ (or in the case of rotating or scanning antennas, 7200 $mW\text{-}s/cm^2$). Also, the maximum permissible average power density shall not exceed 1 mW/cm^2 for 12 minutes.

This new approach by the USSR in establishing permissible microwave exposure levels is in agreement with modern understanding of the dependence of RFR bioeffects on heat load. The proposed changes are apparently based on information developed by the Institute of Occupational Health and Disease of the Professions of the USSR Academy of Medical Sciences.

NATO STANAG 2345

In 1975, Research Study Group 2 on Protection of Personnel Against Non-Ionizing Electromagnetic Radiation (Panel VIII of AC/243 Defence Research Group, NATO) proposed a revision to STANAG 2345. The intent of the proposal was to revise the STANAG to in-

corporate frequency-dependent-RFR safety guidelines. These changes are documented in the current NATO Standardization Agreement (STANAG) 2345 (MED), "Control and Recording of Personnel Exposure to Radiofrequency Radiation." The maximum permissible exposure levels averaged over any 6-minute period for continuous wave, modulated, or pulsed radiation are presented in Table 6 (13).

Table 6
STANAG 2345--Limitation of Exposure for Personnel

Frequency Range	Average Power Density
10 kHz - 1 MHz	265 mW/cm^2
1 MHz - 10 MHz	66 mW/cm^2
10 MHz - 300 GHz	10 mW/cm^2

Figure 4 compares the NATO STANAG 2345, ANSI, IRPA, and USSR standards. Almost all RFR safety guidelines being used today will fall within the range of those presented in Figure 4.

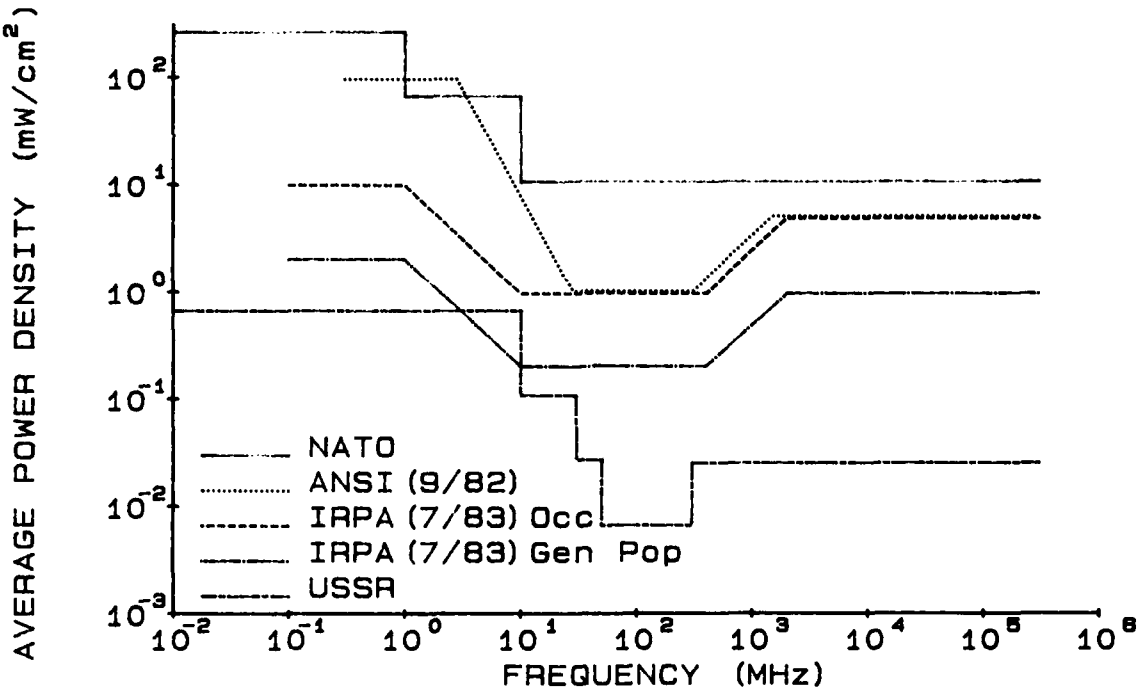

Figure 4. Comparison of RFR safety guidelines

DISCUSSION

Since most RFR standards include some form of time averaging to limit personnel exposures, comparing them on that basis is appropriate. Figures 5 and 6 present the permissible power density in mW/cm^2 versus exposure time in minutes for two frequency bands for several of the standards discussed above. Tables 7 and 8 present the same comparisons, including the SAR and specific absorption (SA) values. Reviewed in this manner, these data illustrate the fact that for relatively short term exposures the differences in current RFR standards used throughout the world are not great.

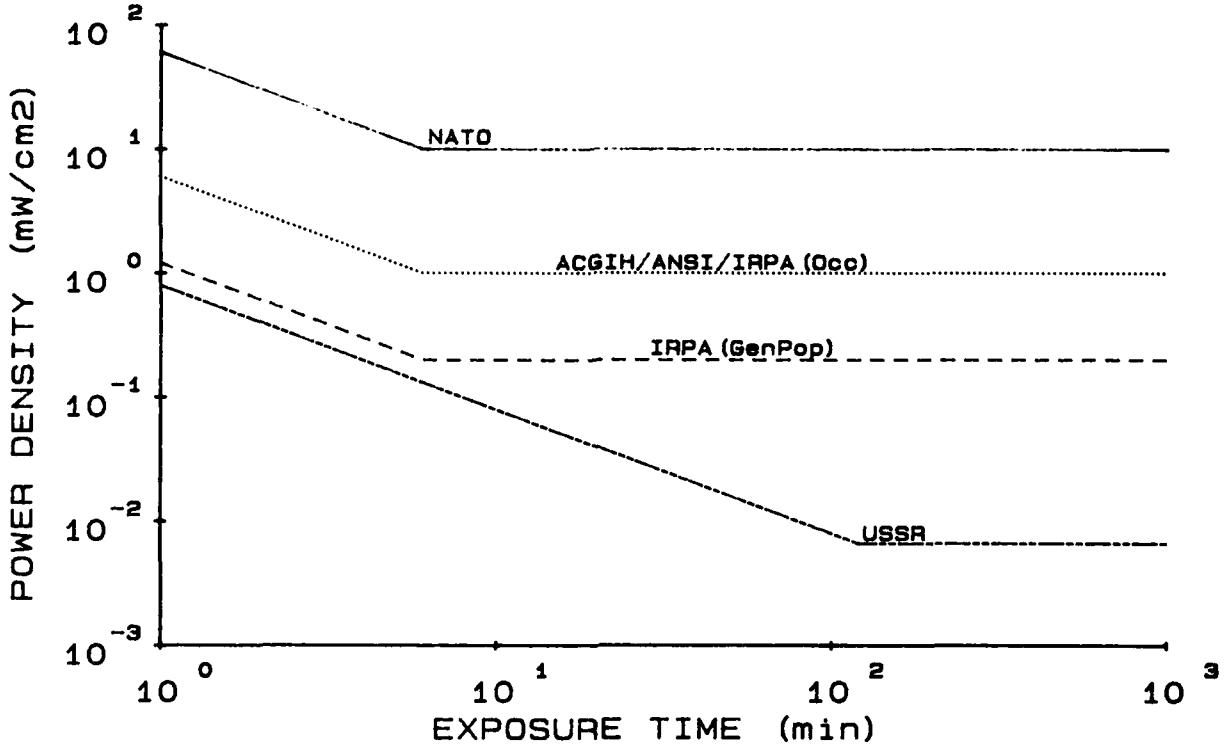

Figure 5. Power density vs exposure time (for frequencies 30 MHz - 2 GHz)

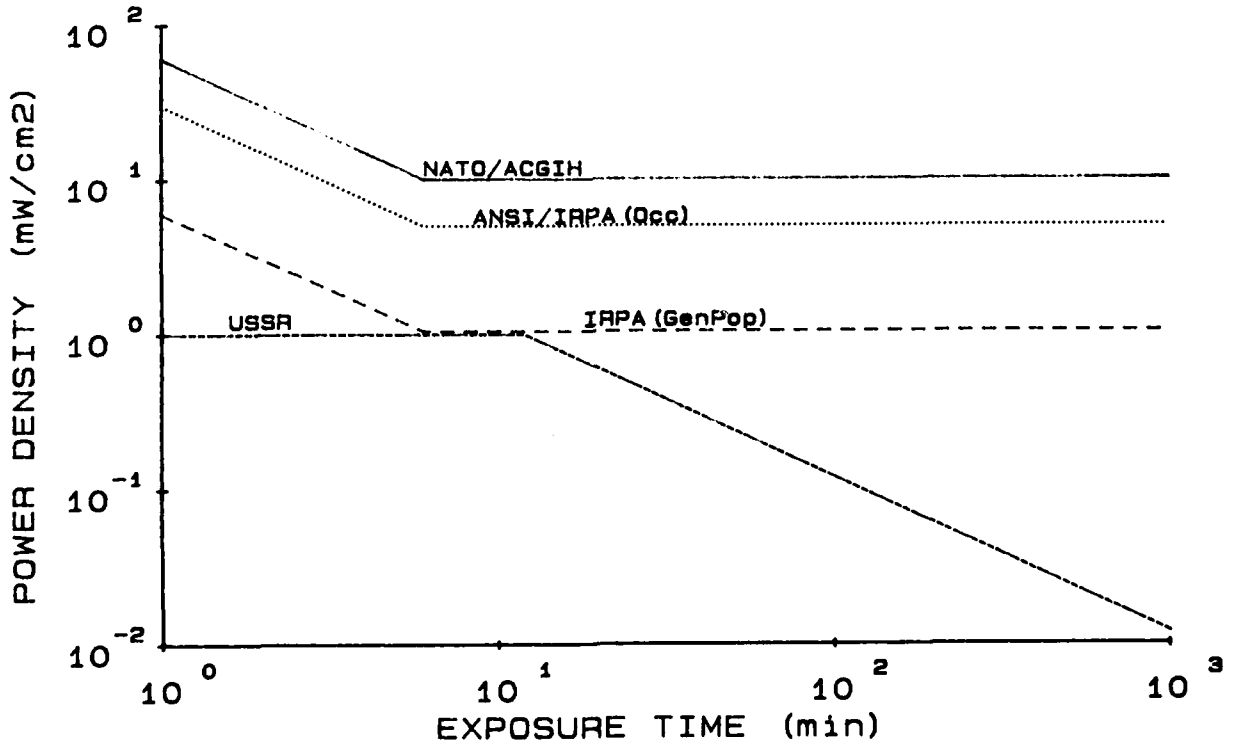

Figure 6. Power density vs exposure time (for frequencies >2 GHz)

TABLE 7
Comparison of RFR Exposure Limits (for Frequencies 30 MHz - 2 GHz)

	Power Density (mW/cm^2)	Exposure Time(H)	Integrated Power Density ($mW-s/cm^2$)	Specific Absorption Rate (SAR) (W/kg)	Specific Absorption (J/kg)
NATO	10	0.1	3600	4	1440
ACGIH	1	0.1	360	0.4	144
ANSI	1	0.1	360	0.4	144
IRPA (Occ)	1	0.1	360	0.4	144
IRPA (GenPop)	0.2	0.1	72	0.08	29
USSR	0.0066	2	48	0.0026	19

TABLE 8
Comparison of RFR Exposure Limits (for Frequencies >2 GHz)

	Power Density (mW/cm^2)	Exposure Time(H)	Integrated Power Density ($mW-s/cm^2$)	Specific Absorption Rate (SAR) (W/kg)	Specific Absorption (J/kg)
NATO	10	0.1	3600	0.4	144
ACGIH	10	0.1	3600	0.4	144
ANSI	5	0.1	1800	0.4	144
IRPA (Occ)	5	0.1	1800	0.4	144
IRPA (GenPop)	1	0.1	360	0.08	29
USSR	0.1	2	720	0.004	29
	1	0.2	720	0.04	29

Safety Considerations

The new RFR exposure guidelines have many built-in safety features that are rarely, if ever, considered. In these days all forms of radiation are often perceived as life threatening, so to highlight some of the inherent safety features associated with the new RFR exposure standards seems appropriate.

SAR Versus Derived Incident Power Density

Many reviews of the RFR bioeffects literature have been written since 1980 (3,8,11,14,15). Based on these reviews, a whole-body SAR of 4 W/kg has often been established as a reasonable value for the adverse effects threshold. Using a 10-fold safety factor, most standards promulgated in 1982-1984 were designed to limit RFR exposures to a whole body SAR of 0.4 W/kg or less. In fact, most of the derived curves for permissible incident power densities provide more than a factor-of-10 safety. For example, the IRPA occupational exposure limit at frequencies above 10 MHz is 0.4 W/kg or less. The derived incident power density in the frequency range 10-30 MHz is set at 1 mW/cm^2; it could, in fact, range from 1 to 9 mW/cm^2, under worst case (physical contact with a ground plane) exposure conditions, without exceeding the 0.4 W/kg whole body SAR. In the resonant region, the ANSI curve is actually closer to 0.35 W/kg than to 0.4 W/kg.

RFR Penetration and Absorption in Biological Systems

The vast majority of biological-effect studies that have been used to establish the current RFR safety guidelines were conducted using small laboratory animals (mice and rats) and 2450-MHz radiation sources. In such studies, the RFR energy is deposited throughout the animal's body and includes "hot spots" of RFR absorption that can be 10 to 20 times higher than the average. The 4-W/kg effects thresholds were established using worst-case data. The most likely human exposures are much less traumatic, because the RFR energy is not generally deposited throughout the body. To illustrate this fact, Table 9 gives some approximations of the penetration depth in biological tissue and the percent of total body mass (in humans) that might be exposed as a function of radiation frequency. Depth of penetration is defined as the distance at which the power absorption is e^{-2} (0.135) of the surface value.

Table 9
RFR Penetration and Absorption in Humans

Frequency of Radiation (GHz)	Depth of Penetration in Tissue (cm)	Percent of Total Body Mass Exposed
1	4.05	20.8
2	2.46	15.6
4	1.66	9.2
8	0.65	4.0
10	0.46	2.9
20	0.16	1.0

For example, a "standard" man exposed to a 1-GHz field might receive a unilateral exposure, penetrating to a depth of ~4 cm and result in ~ 21% of the total body mass receiving RFR energy. For a 10-GHz exposure, the RFR energy might only penetrate to ~0.5 cm and result in less than ~3% of the total body mass receiving RFR energy. In fact, real-world exposure situations are much more complicated because the RFR energy is deposited in a very nonuniform manner, resulting in hot spots which are difficult to predict. These so-called hot spots do not relate to actual temperature excursions but to the fact that the SAR at different locations in the body can vary by more than an order of magnitude. Nevertheless, the data presented in Table 9 illustrate that in most exposure situations the RFR energy is deposited unilaterally in a relatively small volume of the body. In many exposure situations, an appreciable fraction of the body is not subjected to any significant energy deposition for exposures at or below the safety guidelines. This is believed to be an added safety factor when the potential bioeffects of RFR exposures are considered. The body's thermoregulatory system must still handle the total energy deposited, but such heat loads are minimal (less than one half of the basal metabolic rate) for permissible exposure levels.

Guy and Chou (16), using scaled models of a man and a 450-MHz source, measured the SAR and the SAR distribution for 12 possible field polarizations and 4 body postures. Using a 1-mW/cm^2 exposure level, the mean SAR was about 0.05 W/kg and the spatial peak SAR was as high as 0.2, 0.6, and 0.3 W/kg for the neck, wrist, and ankles respectively. These results indicate almost an order of magnitude safety over that designed into the ANSI standard. Additionally, normal thermoregulatory response (blood flow) in living animals minimizes the temperature excursions predicted from static models (17).

Partial Versus Whole-Body Exposures

Most human exposures at radiation intensities approaching the safety limits will result in only a part of the body being exposed. For example, most radar systems propagate radiation beams confined to a few degrees in both directions and depend on scanning to cover the surveillance volume. Human exposures at intensities approaching the maximum permissible values would normally occur only close to the source, where the beam size is relatively small. This situation also exists for exposures to the leakage fields from microwave ovens and from a wide range of RFR-generating equipment. Also, such partial body exposures for frequencies greater than about 1 GHz, at intensities which exceed the normal limits are often felt as a warming sensation, thus warning the person to terminate such an exposure before it becomes more serious.

Subject and Source Dynamics

The radiation protection guides applied over the frequency range covering whole-body resonant conditions were selected to protect the human under the worst circumstances (1,2,3,4). They assume the human would be exposed for 6 minutes to a free-space plane-wave field at the radiation frequency equivalent to his or her resonant frequency (dependent on the person's height), with the electric field vector aligned with the long axis of the body, and at the maximum RFR radiation intensity allowed by the guideline. These circumstances seldom, if ever, occur. For example, it is rare that a person would be in a field having his or her resonant frequency at the maximum intensity allowed. Even with a measured level equal to the maximum, the isodose intensity contour would not likely be as large as the human. Also, it is unlikely that the person would maintain an erect posture, particularly for 6 minutes at a time. Changes in posture such as stooping, bending, or squatting significantly reduces the RFR energy absorption. This point is illustrated in Figure 7, where relative power absorption curves have been plotted for a 1.8-m, 70 kg human who has changed his or her effective height by squatting, sitting, standing with arms in normal position, and raising the arms above the head. As in Figure 1, these curves were developed using prolate spheroid models with a constant body mass of 70 kg. In some jobs people might remain a fixed distance from an RFR source, but in most exposure situations there is considerable movement between the source and subject. Such movement often reduces the amount of RFR energy absorbed.

Considering the above facts, some significant, albeit unquantified, additional safety factor is inherent in the application of these new guidelines.

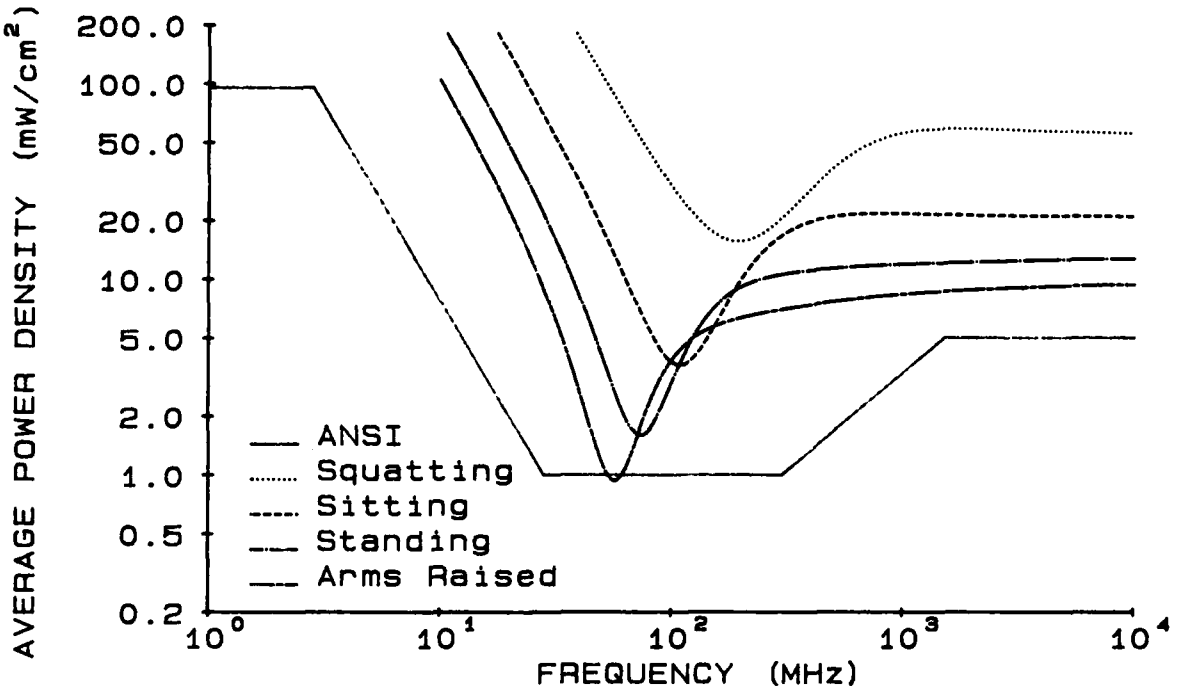

Figure 7. Power densities that limit human whole-body SAR to 0.4 W/kg for a 1.8-m person

CONCLUSIONS

Advancements in understanding RFR interactions with living systems, based on dosimetry and biological-effect research, are shared throughout the world. Differences in RFR safety guidelines established by different governmental bodies depend largely on the degrees of conservatism applied and philosophical approaches taken. These facts are well documented in current reviews of RFR-induced biological effects (3,4,8,15).

New research to assess biological effects of RFR exposure has emerged rapidly and in considerable quantity over the past 5 years. The vast majority of reported effects are related to thermal insults from SARs greater than 4 W/kg. The new RFR safety guidelines and their rules of application provide greater safety than those used in the past. They have a more credible scientific basis and are supported by the majority of current research findings.

FUTURE TRENDS IN RFR STANDARD SETTING

The acceptance of whole-body averaged SAR in the development of the new frequency-dependent-RFR safety standards has been a significant improvement. It relates well to laboratory studies using small animals subjected to fields at frequencies close to resonant conditions.

Despite these facts, whole-body SAR is not an adequate basis for the RFR safety guidelines at frequencies greater than 20 GHz and less than 3 MHz. At frequencies greater than 20 GHz, RFR energy deposition in biologic tissue is very superficial, as shown in Table 9, and some form of localized SAR would serve as a better safety guideline than a whole-body averaged SAR. New safety guidelines for the 20-300 GHz frequency range will likely emerge in the next few years.

Also, whole-body averaged SAR is not an adequate basis for the safety guidelines below ~3 MHz since the RFR absorption in biologic tissue decreases as a function of frequency squared below the resonant point. The actual RFR energy absorption becomes so small in this frequency range (10 kHz - 3 MHz) that it can be considered safe at any practical value of incident power density. Valid questions remain, however, concerning the potential for shocks and burns. These questions are being studied under

current research programs, and new safety guidelines can be expected in the next couple of years. Interim guidelines to minimize shock and burn are contained in the ACGIH TLV standard.

In summary, for the frequency range of greatest concern to present military RFR operations (3 MHz - 20 GHz), current safety guidelines based on whole-body averaged SAR are appropriate. Some refinements can be expected, however, as dosimetric methodology is improved and biological effects are better defined.

ACKNOWLEDGMENTS

Special appreciation is extended to Mr. William D. Hurt, Radiation Physics Branch, Radiation Sciences Division, *U.S. Air Force School of Aerospace Medicine*, for developing the information used in the tables and figures of this paper. We are also indebted to First Lieutenant Luis G. Lozano (deceased) for developing the computer programs used to generate the data base from which much of this information was selected.

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Radio Frequency Radiation (RFR) Measurements in Operational Settings

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Summary

This is a detailed discussion of the principles, procedures, and instrumentation required to carry out routine RFR measurements under field or operational conditions. The information herein contained comes largely from several years of experience gathered by the USAF Occupational and Environmental Health Laboratory (USAF OEHL) and its predecessor the USAF Radiological Health Laboratory (USAF RHL). There are few literature references available in this area and much of the information is based on a previously published report (1) and first-hand experiences.

Preliminary Considerations

The technology to make routine meaningful, reliable, and repeatable radio frequency radiation (RFR) power density measurements in the field is now readily available. Over the last 12 years there have been significant advances in power density instrumentation technology that now provide us with lightweight portable devices that exhibit acceptable accuracy. The development and evolution of that instrumentation was largely driven by requirements that the U.S. Air Force first defined in the early 1970s.

Prior to 1972, field power density instruments were essentially limited to RAMCO 1200 and Empire Devices NF-157 instruments that employed horn-type antennae which manifested serious deficiencies including a total inability to accurately capture RFR travelling in a circularly polarized mode. The most glaring deficiency, however, was the fact that standard gain horns, or similar antennae, are incapable of accurately dealing with Fresnel or near field wave fronts. Experience over the years has shown that a large percentage of all personnel hazards from RFR emitters lie in the near field. Therefore, instruments that utilize horn-type antennae have serious limitations.

In the spring of 1972, the Narda Microwave Corporation introduced to the market the first "user friendly" isotropic broadband power density devices that were to revolutionize the making of field measurements. Those early instruments were only capable of measuring average power densities up to 20 mW/cm^2 with a 60 $Watt/cm^2$ peak power burnout rating. First attempts to use these early instruments (Model 8300), in the investigation of an alleged overexposure, were disastrous. The emitter in question was an FPN-16 Precision Approach Radar with an average power output of approximately 40 watts in the X-band. The duty factor (DF), however, was very short; e.g., 0.000339. That short DF was capable of causing peak powers considerably in excess of the 60 Watt limit. It was because of this peculiarity that the first two 8300 probes were burned out within a matter of seconds, even though the average power density encountered never exceeded 6 mW/cm^2. This phenomenon was not adequately explained for several weeks and determination of the cause led directly to Narda developing a series of increasingly hardened probes. Today, state-of-the-art probes exist that are capable of withstanding several $Watts/cm^2$ average power density, and several hundreds of $Watts/cm^2$ peak power density.

Even the early isotropic probes exhibited essentially flat responses; i.e., $\pm$ 0.5 dB, over a frequency range of 300 MHz to 18 GHz and measured E-field equivalent average power density. These devices utilized orthogonal dipoles that rendered them at least theoretically isotropic, which provided an accuracy capability in the near field not previously possible. Many improvements have since been made so that reliable E- and H-field measurements are now possible from 300 KHz to more than 34 GHz.

Over the last 12 years the USAF OEHL and its predecessor organization have evaluated and field tested a wide variety of RFR power density instrumentation designed, developed and manufactured by a number of firms and agencies. Each of these instruments has some outstanding characteristics and some equally poor features. At the present time the USAF OEHL inventory of power density instrumentation is made up primarily of Narda Microwave devices.

Making reliable RFR field measurements is often very difficult. Modern isotropic equipment has overcome many of the earlier problems such as temperature sensitivities, static interferences, and voltage fluctuations. The physical and organizational problems in conducting RFR field surveys shall no doubt always be present.

It is our opinion, based on a number of years of experience, that only about 15% of the RFR emitters will account for about 95% of the measurement problems. There are a number of classes of emitters that may be easily and promptly dismissed from consideration as a potential hazard to personnel. For instance, hand-held transceivers, commonly known as "bricks", which operate from 136 to 174 MHz and in the 510 MHz regions of the spectrum are considered to be nonhazardous to personnel if they emit less than 7 watts, as virtually all do. As another example, many large very high-powered emitters have main beams that are not normally accessible to personnel. Whatever levels such emitters may generate are

largely of academic interest only, because personnel simply cannot get to the hazard during the normal course of day-to-day activities. In general, emitters that utilize very thin horizontal or vertical antennae with essentially omni-directional patterns have personnel hazards that are very easily managed by applying the results of many previous measurements that have been used to validate the data depicted in Table 1, and observing the caveats that are noted. Finally, many of the older and very common emitters have been measured over and over again. Such data are summarized for the Air Force at USAF OEHL and are readily available to the field. The U.S. Army and Navy have somewhat similar counterpart organizations where this kind of data for their respective emitters are maintained for reference. By contacting one or more of these sources, at least some information regarding virtually every emitter operated by the U.S. military should be available.

Table I

Personnel Hazard Predictions for Thin Vertical and Horizontal Omni-Directional Antennae (2)

Transmitter Power In Watts	Vertical						Horizontal			
	10 mW/cm²		1 mW/cm²		0.1 mW/cm²		10 mW/cm²		1 mW/cm²	
	Feet	Meters	Feet	Meters	Feet	Meters	Feet	Meters	Feet	Meters
10	0.7	0.20	2.1	0.64	5.2	1.59	0.4	0.13	1.4	0.43
20	0.8	0.25	2.6	0.79	7.1	2.17	0.6	0.18	1.9	0.58
30	1.0	0.30	3.1	9.46	8.9	2.71	0.7	0.22	2.3	0.70
40	1.2	0.36	3.7	1.13	10.6	3.23	0.8	0.25	2.6	0.79
50	1.3	0.40	4.2	1.28	14.7	4.48	0.9	0.28	2.9	0.88
75	1.6	0.50	5.2	1.59	16.4	5.00	1.1	0.35	3.6	1.10
100	1.8	0.56	5.8	1.77	18.6	5.67	1.3	0.40	4.2	1.28
120	2.0	0.62	6.4	1.95	20.1	6.13	1.4	0.44	4.6	1.40
150	2.3	0.70	7.3	2.23	22.8	6.95	1.6	0.49	5.1	1.56
200	2.6	0.80	8.3	2.53	--	--	1.8	0.56	5.8	1.77
250	3.0	0.90	9.3	2.84	--	--	2.0	0.63	6.5	1.98
400	3.9	1.20	12.5	3.81	--	--	2.6	0.80	8.3	2.53
500	4.1	1.26	13.1	4.00	--	--	2.9	0.89	9.2	2.81
750	4.9	1.50	15.6	4.76	--	--	3.6	1.10	11.4	3.48
1000	5.8	1.78	18.5	5.64	--	--	4.1	1.26	13.1	4.00
1500	7.2	2.20	22.8	6.95	--	--	5.0	1.55	16.1	4.91
2000	8.2	2.50	25.9	7.90	--	--	5.8	1.78	18.5	5.64

Notes:
1. Predictions may be applied to omni-directional antennae with gains of 6 dB or less.
2. Table may be applied to frequencies between 3 and 600 MHz.
3. Although these data do not represent a linear relationship, interpolation is possible, but will cause the distances to be even more conservative.

Prior to making measurements, all available information on the RFR emitter should be gathered and studied. It is an essential first that the nominal operating characteristics of the emitter in question be known. This is often much more difficult than you might expect. As a minimum, the following emitter information must be obtained before proceeding with any survey:

1. Operating Frequency
2. Peak Power
3. Pulse Width (PW), if any
4. Pulse Repetition Frequency (PRF), if any
5. Antenna Gain in dB
6. Antenna Dimensions
7. Beam Width (BW)
8. Scan or Rotation Rate, if any

You will often find that some of this information is difficult to obtain. It is unfortunate, but true, that operators of RFR emitting equipment often do not know all and sometimes none of the parameters of their emitter and, they frequently don't know where to find the data. Many times you will spend hours and hours getting ready to do a survey that will require only minutes and much of that time will often be consumed getting the nominal characteristics properly identified. There is, unfortunately, no simple and foolproof solution to this particular problem, and it frequently challenges the most ingenious of surveyors. If the emitter operators cannot provide the information, the first place to look should be the applicable Technical Order (T.O.), which in many cases still may not contain everything. Even if the operators do supply you with the data, you must never assume that it is totally accurate. Therefore, an alternate data source for confirmation is most helpful. Those alternate sources might even include the emitter manufacturer.

By whatever means possible, you will eventually obtain the necessary information and the next step is to estimate the hazard distance in order to give you a starting place for making measurements. If this is not done, it is quite easy to expose oneself to unnecessarily high levels of RFR at the outset of your survey. There are three possible and practical ways to estimate the hazard:

1. Previous survey results
2. Computer modeling
3. Far field calculations (Inverse Square Law)

The first is, of course, the best and usually the easiest, if previous surveys have been done on the emitter in question. The second method can yield very accurate results, but there are only a few agencies that have the capability. The parameters needed for a computer model are the same as those already noted. Not all emitters can be acceptably handled, the limitation being what sort of illumination is employed. The third method may be thought of as a "quick and dirty" one that is useful and yields very conservative results. It assumes that plane-wave, far-field conditions exist in close to the antenna though that is hardly ever the case. The only data needed are the peak power, PW, PRF, antenna gain in absolute terms, and the hazard level you are interested in. By multiplying the PW by the PRF the duty factor (DF) is obtained and when the peak power is multiplied by that DF one obtains the average power (Pav). Since antenna gains are specified in dB, which is a logrithmic expression, it is necessary to convert them to a "multiplication factor" by use of the following equation:

$$G\ abs = \text{anti/log}\ (G\ db/10)$$

Table II is much easier to use however, and once the necessary conversions have been made and the average power obtained, the following equation can then be employed to calculate the distance.

$$D = \sqrt{\frac{(\text{Power})\ (\text{Gain})}{4\ \pi W}}$$

Where: D = distance in meters
W = power density of interest in Watts/cm²

For 10 mW/cm², the equation then is:

$$D\ (10\ \text{mW/cm}^2) = \sqrt{\frac{(\text{Pav})\ (G\ \text{abs})}{4\ \pi (100)}}$$

D will be in meters
Pav must be in Watts

This equation will provide you with a very conservative estimate of the hazard distance, which you will find useful as a starting point for your measurements. The factor of conservatism will vary from as little as one, to as great as 5.0, depending on how far from the antenna the far field really begins. There are some small aperture antennae operating in the J- and X-bands that have short near fields where this equation will yield very accurate predictions. The important point to remember is that <u>any</u> method other than actual measurements is only an estimate and/or prediction, and all are markedly influenced by a variety of factors, most of which are unknown or poorly understood. Actual measurements are always preferable, but estimates are useful as tentative numbers and for a starting place for any survey.

Table II

Antenna Power Gain--Conversion Between Absolute and Decibel Units

Gain in dB	Absolute Gain	Gain in dB	Absolute Gain	Gain in dB	Absolute Gain
1.0	1.26	20.0	100.00	40.0	10000.00
1.5	1.41	20.5	112.20	40.5	11220.18
2.0	1.58	21.0	125.89	41.0	12589.25
2.5	1.78	21.5	141.25	41.5	14125.38
3.0	2.00	22.0	158.49	42.0	15848.93
3.5	2.24	22.5	177.83	42.5	17782.79
4.0	2.51	23.0	199.53	43.0	19952.62
4.5	2.82	23.5	223.87	43.5	22387.21
5.0	3.16	24.0	251.19	44.0	25118.86
5.5	3.55	24.5	281.84	44.5	28183.83
6.0	3.98	25.0	316.23	45.0	31622.78
6.5	4.47	25.5	354.81	45.5	35481.34
7.0	5.01	26.0	398.11	46.0	39810.72
7.5	5.62	26.5	446.68	46.5	44668.34
8.0	6.31	27.0	501.19	47.0	50118.72
8.5	7.08	27.5	562.34	47.5	56234.13
9.0	7.94	28.0	630.96	48.0	63095.73
9.5	8.91	28.5	707.95	48.5	70794.58
10.0	10.00	29.0	794.33	49.0	79432.82
10.5	11.22	29.5	891.25	49.5	89125.09
11.0	12.59	30.0	1000.00	50.0	100000.00
11.5	14.13	30.5	1122.02	50.5	112201.85
12.0	15.85	31.0	1258.93	51.0	125892.54
12.5	17.78	31.5	1412.54	51.5	141253.75
13.0	19.96	32.0	1584.89	52.0	158489.32
13.5	22.39	32.5	1778.28	52.5	177827.94
14.0	25.12	33.0	1996.26	53.0	199526.23
14.5	28.18	33.5	2238.72	53.5	223872.11
15.0	31.62	34.0	2511.89	54.0	251188.64
15.5	22.39	34.5	2818.38	54.5	281838.29
16.0	39.81	35.0	3162.28	55.0	316227.77
16.5	44.67	35.5	3548.13	55.5	354813.39
17.0	50.12	36.0	3981.07	56.0	398107.17
17.5	56.23	36.5	4466.84	56.5	446683.59
18.0	63.10	37.0	5011.87	57.0	501187.23
18.5	70.79	37.5	5623.41	57.5	562341.33
19.0	79.43	38.0	6309.57	58.0	630957.34
19.5	89.13	38.5	7079.46	58.5	707945.78
		39.0	7943.28	59.0	794328.23
		39.5	8912.51	59.5	891250.38
				60.0	1000000.00

Instrumentation

At this point you have established, by whatever means, a starting place to make your measurements and are now just about ready to take the instruments out of the case. It is assumed for purposes of this paper that the measurements will be made using Narda 8600 Equipment and that the power density level of interest is 10 mW/cm². Further, in order to describe the worst of all possible situations, the emitter to be evaluated is of a pulsed, i.e., radar, variety.

Narda 8600 E-field probes are available in two power handling types. The 8621 is white in color and will handle up to 60 mW/cm² average power density and 60 Watts/cm² peak power density. The 8623 probe is yellow and will handle up to 300 mW/cm² average and 300 Watts/cm² peak power. Both probes are normally calibrated at several points over the 300 MHz to 26 GHz frequency range. The larger 8616 meter accomplishes the coverage of the 30 dB dynamic power density range through the use of a 3 position (range) switch. A smaller and much more portable 8601 meter is available, but lacks many of the more sophisticated features of the larger and heavier 8616.

For field measurements, these 8620 series probes may be considered to be isotropic and, if used properly, are quite capable of yielding accurate, reliable, repeatable and very useful power density data. As an aside, Narda also makes several other probe series for measurements to as low as 300 KHz.

All Narda probes are susceptible to burnout when exposed to high power densities. Be aware that these probes can be burned out even though they are not connected to a meter. Probe burnout is generally not a problem when making measurements of continous wave (CW) emissions, because the burnout threshold is much higher than the maximum meter reading. Under CW conditions, as long as the surveyor does not allow the meter to exceed full scale deflection (20 mW/cm² for white and 100 mW/cm² for yellow probes), the risk of burnout is negligible. However, when measurements of pulsed emissions are undertaken, the risk of burnout becomes much greater and is inversely proportional to the length of the DF; e.g., the shorter the DF the greater the risk. If when the DF is short enough, the <u>average</u> power emanating from an emitter may be quite low while the

peak power can be quite high, and the peak power absorbed by the probe may easily exceed the peak overload value. The probe will then fail even though the average power indicated by the meter is something less than the full scale reading. The following equation can be used to avoid a rather costly accident:

$$PD_{max} = DF \times BR/CF$$

Where: PD_{max} = Maximum meter reading before probe burnout will occur.

DF = Duty factor of emitter being evaluated.

BR = Probe burnout rating (e.g., 3×10^{4} mW/cm² for yellow probes, 6×10^{4} mW/cm² for white probes).

CF = Probe correction factor at the frequency being measured.

If PD_{max} exceeds 100 mW/cm² for yellow probes or 20 mW/cm² for white, there is no cause for any special concern on the part of the surveyor regarding probe failure, as long as the meter is never allowed to go off scale at the high end. On the other hand, if PD_{max} turns out to be less than the maximum power density ratings of the probes being used, the surveyor must be very very careful not to allow the meter to exceed the PD_{max}. Even brief excursions will almost certainly burn the probe out. The following examples should help clarify this matter:

Example 1: PW = 2 µsec
PRF = 360 Hz
DF = 0.00072 (PW x PRF)
CF = 1.2 for a 8623 (yellow) probe

PD_{max} = 0.00072 x 300000/1.2
= 180 mW/cm² which is greater than a full scale meter reading.

Conclusion: Low risk of probe failure if meter needle is kept on scale

Example 2: PW = 0.25 µsec
PRF = 800 Hz
DF = 0.0002
CF = 0.95 for a 8623 (yellow) probe

PD_{max} = 0.0002 x 300000/0.95
= 63 mW/cm² which is nearly 37% less than a full scale meter reading

Conclusion: Very high risk of probe failure. Be very careful and never allow the meter reading to exceed approximately 60 mW/cm²

Final Presurvey Considerations

In preparation for field measurements you have now completed nearly all of the preliminaries and are almost ready to begin the measurement phases. There will be instances where you will not have and cannot get the necessary instrumentation to make measurements, usually because of the frequency of the emitter. These cases will almost always involve emitters operating at frequencies below 30 MHz, sometimes much below. Such instances lie outside the scope of this paper.

The next-to-last step before making measurements is to check out the equipment. The burnout threshold problem has already been discussed and all that remains is to make certain the meter battery voltage is within tolerance and the probe(s) are within the manufactures calibration interval(s).

The final preliminary step is to consider the safety aspects of what you are about to do. The only successful survey is a safe survey which produces the necessary data and results in the understanding and satisfaction of all those involved. This is usually not as easy to accomplish as it may seem. Consider the following matters before you begin:

1. Completely brief all involved personnel on exactly what you are going to do, how it will be done, and what you specifically wish to accomplish. You must inspire confidence.

2. Establish an absolutely positive and fail-safe communications link between yourself and the operator of the emitter. A most important part of this is the assumption that you, the surveyor, have absolute control over the emitter during the survey.

3. Always begin your measurements at a distance greater than where the hazard is expected to be.

4. Remember that surveyors must not subject themselves to an overexposure.

5. Anticipate unexpected problems and be flexible in your approach.

For those of you who appreciate the value of a checklist and are more comfortable with one, the following has been developed over the years.

RADIO FREQUENCY RADIATION SURVEY CHECKLIST

I. PRE-SURVEY PHASE

A. Contact person(s) in charge; obtain and record:

1. Exact location of emitter
2. Description of emitter environment
3. Names, office symbols, and extensions of persons who are knowledgeable and/or responsible
4. Emitter operating parameters

B. Coordinate arrangements for the survey:

1. Date and time when emitter will be available
2. Personnel to operate the system
3. Mobile lifting equipment, climbing gear, etc., as required
4. Miscellaneous support items

C. Perform calculations:

1. Estimated hazard distance
2. Probe burnout level
3. Probe correction factor

D. Check equipment:

1. Battery levels
2. Probe and meter function
3. Calibration due date

II. SURVEY PHASE

A. Contact person in charge; inbrief as necessary

B. Arrange for emitter set-up in "worst-case" mode

C. Using correct technique, locate and record (if practical):

1. PEL hazard radius and height above ground
2. All areas in which the PEL could be exceeded
3. Levels at work stations and "normally accessible" areas
4. Any "hot-spots"

D. Observe and note:

1. Adequacy of warning signs and access-limiting devices
2. Adequacy of any standard procedures used to reduce or avoid exposure to radiation
3. Degree of caution exercised by workers
4. Knowledge of workers about handling a suspected overexposure

E. Outbrief as necessary

III. POST-SURVEY PHASE

A. Analyze results; formulate conclusions and recommendations.

B. Prepare letter/report for concerned offices.

C. File data, photographs, drawings, correspondence, etc., in shop folder.

RFR Emitter Identification and Nomenclature

In the United States, most emitters have standard nomenclatures assigned to them according, more or less, to a logical scheme. The so called "AN" nomenclatures consist of three letters, a dash and one, two or three numbers. The letters have meaning and generally describe what the emitter's primary function is according to Table III.

The three numbers that follow the letter have little or no meaning to measurement personnel in that they are indicators of chronological developmental or generational sequence only.

While most emitters have "AN" designations, <u>some</u> do not and it is quite easy to overlook a potentially hazardous emitter because its designation does not conform to the "AN" scheme.

Table III

"AN" Nomenclature Scheme (3)

First Letter How Installed	Second Letter Type of Equipment	Third Letter General/Primary Purpose
A - Piloted Aircraft	A - Invisible Light, heat radiation	A - Auxiliary assemblies (not complete operating sets used with or part of two or more sets series)
B - Underwater mobile, submarine	B - Pigeon (do not use)	B - Bombing
C - Air transportable (inactivated, do not use)	C - Carrier	C - Communications (receiving and transmitting)
D - Pilotless carrier	D - Radiac	D - Direction finder, reconnaissance, and/or surveillance
F - Fixed Ground	E - Nupac	E - Ejection and/or release
G - General ground use	F - Photographic (not used in U.S.)	G - Fire-control, or searchlight directing
K - Amphibious	G - Telegraph or teletype	H - Recording and/or reproducing (graphic meteorological and sound)
M - Ground, mobile	I - Interphone and public Address	K - Computing
P - Portable	J - Electromechanical or Inertial wire covered	L - Searchlight control (inactivated, use G)
S - Water Surface	K - Telemetering	M - Maintenance and/or test assemblies (including tools)
T - Ground, transportable	L - Countermeasures	N - Navigational aids (including altimeters, beacons, compasses, racons, depth, sounding approach, and landing)
U - General utility	M - meteorological	P - Reproducing (inactivated, use H)
V - Ground, vehicular	N - Sound in air	Q - Special, or combination of purposes
W - Water surface and underwater combination	P - Radar	R - Receiving, passive detecting
Z - Piloted and pilotless airborne vehicle combination	Q - Sonar and underwater sound	S - Detecting and/or range and bearing, search
	R - Radio	T - Transmitting
	S - Special types, magnetic, etc., or combinations of types	W - Automatic flight or remote control
	T - Telephone (wire)	X - Identification and recognition
	V - Visual and visible light	
	W - Armament (peculiar armament, not otherwise, covered	
	X - Facsimile or television	
	Y - Data processing	

Survey of Ground-Based RF Emitters

Ground-based RF emitters generally bear "AN" nomenclatures beginning with the letter F, M, G, or T, denoting the following:

a. Fixed

FPN-47	RAPCON (Radar Approach Control)
FPS-90	Height finder
FRT-49	Ground to Air Communications

b. Mobile

MPN-14	Area Surveillance and GCA (Ground Control Approach)
MRC-113	High powered tropospheric scatter communications unit
MPS-9	Area Surveillance and Guidance

c. Ground

GPN-12	State-of-the art RAPCON
GRN-20	TACAN (Tactical Air Control and Navigation)
GRC-75	Flight Facilities

d. Transportable

TPS-43	Tactical Air Control Radar
TRC-97	Medium powered tropospheric communications unit
TPB-1	Threat simulator

Various modifications of a given emitter may have a letter (A, B, C, etc.) following the numbers.

Ground mounted radar systems are sometimes capable of operating in more than one mode. It is, therefore, vital that during the presurvey, careful consideration be given to all of the possible modes to insure that measurements will be made with the system operating in the mode which will create the "worst case" (highest peak power, highest duty factor, and narrowest beam configuration).

A visual inspection of the site should be made to determine if the main radiated beam is normally accessible to personnel. If not, then there is no hazard, but it must be recognized that there may be future modifications of either the emitter itself or the environment that may make the beam accessible.

If the main beam is normally accessible to personnel, antenna rotation (if applicable) must be stopped and access to the main beam gained at a distance from the antenna determined during presurvey. The beam size, shape, and character should be determined, then the actual limit of the appropriate personnel hazard distance located. In order to assure that meter readings are accurate, care must be taken to keep the probe handle parallel to the beam axis, or perpendicular to the emitter surface as appropriate. In addition, try to avoid beam reflections from nearby objects.

Regardless of whether or not the main beam is normally accessible, the area surrounding the antenna itself should be carefully probed for possible hazardous levels of energy, as well as a determination made as to what might be required for personnel to access hazardous levels in the immediate vicinity of the antenna proper.

WARNING: When surveying aperture type systems, the area between the feedhorn and the reflector is normally very dangerous, both to personnel and to the RF power density probes, and should be very carefully avoided by both.

Operating personnel should be asked to accurately determine the actual power input value at the time measurements were made. Many ground systems have integral directional couplers and power meters available for this purpose.

An inspection should be conducted to determine if the system under evaluation has adequate interlock mechanisms, and to ascertain if they can be or are, in fact, regularly bypassed for routine maintenance or other purposes.

A visual inspection should be made to determine if there are appropriate RF warning signs in sufficient numbers, and at proper locations.

Operating and maintenance personnel should be interviewed relative to their acquaintance with the potential health hazards associated with radio frequency emissions. It is often possible to gain further insight into this area by observing the activities of these personnel as they go about their normal activities. Technical Orders for each emitter being evaluated should be reviewed for the presence and adequacy of warnings to personnel regarding these hazards. It should also be determined if there are, in fact, adequate, up-to-date written operating and accident reporting Standard Operating Procedures (SOPs) that provide acceptable personnel protection.

Survey of Airborne RF Emitters

Airborne RF systems usually bear "AN" nomenclatures beginning with the letter A. Some examples include:

a. APQ-100, 109, and 120: All fire control systems on the various Air Force versions of the F-4

b. APN-59: A navigational and weather radar common to many aircraft including the C-141, most models of the C-130 and the KC/KB-135

c. APQ-128: Terrain Following Radar (TFR) aboard all models of the F/FB-111

d. ASG-21: Search/tracker aboard the B-52 G & H.

As with ground systems, it is usual to suffix the nomenclature with a letter to designate various modifications or technical updates.

There are several airborne systems with atypical or "non-AN" nomenclatures, such as:

a. MD-9: Search/tracker aboard the B-52 D

b. R-14C: Navigational/weather radar aboard some models of the C-140 and T-39

c. Multimode: the dual band navigational/weather radar aboard the C-5A

When airborne systems are fired live on the ground, the main beam is almost always normally accessible to personnel, and the possible hazards must be recognized by both operating and survey personnel prior to actual measurements.

Airborne antennae, in general, and RADAR antennae, in particular, are often at or very near eye level above the ground, and it must be recognized that, in the normal course of operation, the main beam is often directed downward.

Airborne RADAR are very often capable of operating in many different modes. It is, therefore, vital that an adequate presurvey analysis be accomplished to insure that measurements will be made with the system operating in the mode which will create the "worst case" (highest average power output and narrowest beam widths).

When surveying airborne systems, it is essential that the aircraft be positioned with an ample clear area in front of the antenna to preclude unnecessary radiation of other aircraft, vehicles, buildings, etc. This distance should be determined during the presurvey. The antenna should be stopped and positioned dead ahead in azimuth, and at zero degrees or slightly above in elevation. This last point is necessary in order to prevent reflections from the ground which can create unwanted, unpredictable, and possibly dangerous "hot-spots".

The antenna should be approached from a known safe distance and the main beam located. Once found, its size, shape, and other characteristics should be determined, then the antenna approached until the appropriate hazard distance is located. Care must be taken to maintain the probe handle parallel to the main beam axis.

The area immediately surrounding the antenna (to the side and behind) should be probed for hazardous side lobes and back scatter. These are not commonly seen. As with ground aperture systems, the area between the feedhorn and the reflector is very dangerous and should be avoided by both the RF probe and personnel.

It is highly desirable to evaluate a minimum of three different transmitters (three different aircraft) of a given emitter. In addition, actual power input values should be obtained from operating personnel if at all possible. Many airborne systems have integral directional couplers for this purpose.

The potential for personnel RF hazards in the repair and maintenance (avionics) shops is very great. Most systems are ordinarily fired only into dummy loads in the shops, but some require actual radiation through an antenna. In the former case, the dummy loads should be evaluated for effectiveness, and in the latter case, the evaluation should be similiar to that of the aircraft mounted system, and should include a careful evaluation for possible reflections and scattering within the shop area. An inspection should be conducted to make certain that the area immediately in front of any radiating antenna is off limits to personnel, vehicles, etc., to a distance appropriate for the emitter.

The shop area should be inspected for the presence of appropriate warning signs (if warranted) in sufficient numbers and at appropriate locations.

Both operating and maintenance personnel should be interviewed relative to their acquaintance with the potential health hazards associated with radio frequency emissions. In addition, it will be useful to observe their activities in both the shop and flight line environments in order to gain some feeling for the prevailing attitudes regarding these hazards.

Technical Orders for each emitter being evaluated should be reviewed for the presence and adequacy of warning to personnel regarding radio frequency hazards. It should also be determined if there are, in fact, adequate, up-to-date written operating and accident reporting procedures that provide acceptable personnel protection.

During flight line measurements, observations should be made to determine if there are adequate and effective procedures to protect personnel during routine ground firing of these systems.

Survey of Medical RF Emitters

The most common medical RF emitter is the diathermy machine. These units can usually be found in the physical therapy section of many hospitals and clinics. Medical diathermy machines in the U.S. are authorized to operate on a number of frequencies, but by far the most common are 13.56 and 27.12 MHz (short wave diathermy) and 2450 MHz (microwave diathermy). Most units within the Air Force Medical Service operate on the two lower frequencies. A definitive study has been accomplished on these units and copies of the report were sent to all USAF hospitals.

The prime concern in evaluating diathermy units is NOT with the patient undergoing treatment, since it is assumed the therapy is being administered by or under the supervision of competent professional personnel. There is a potentially significant hazard to the operators of this equipment, particularly the S-band units (2450 MHz). Evaluation may be necessary to be assured that the therapists operate the equipment in a manner that will not cause them to be unnecessarily exposed, particularly to the head and shoulders. (Note: The proper probe for measuring radiation from shortwave diathermy units is not available at most bases.)

Post Survey

The data you have gathered must now be analyzed and some conclusions drawn. Those conclusions should then logically generate certain recommendations or suggested actions. Regardless of the purpose, some sort of written document must always be prepared that will preserve the data for whatever future use might be dictated.

Field measurements of RFR emitters are not difficult today, primarly because of the ready availability of reliable, portable, and acceptably accurate instrumentation. With a little forethought and adequate planning and preparation very useable data can be obtained.

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Acknowledgements

The author would like to thank Mr John Mitchell, USAFSAM/RZP, for his encouragement and technical advice, 1Lt Burl Olsen, USAF OEHL/RZN, for sharing his engineering expertise, and Major Ed Maher, USAF OEHL/RZN, for insuring the accuracy of the procedures herein contained.

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REPORT DOCUMENTATION PAGE

1. Recipient's Reference	2. Originator's Reference	3. Further Reference	4. Security Classification of Document
	AGARD-LS-138	ISBN 92-835-1494-7	UNCLASSIFIED

5. Originator Advisory Group for Aerospace Research and Development
North Atlantic Treaty Organization
7 rue Ancelle, 92200 Neuilly sur Seine, France

6. Title THE IMPACT OF PROPOSED RADIO FREQUENCY RADIATION STANDARDS ON MILITARY OPERATIONS

7. Presented on 11—12 April 1985 in Rome, Italy, 15—16 April 1985 in Lisbon, Portugal and 18—19 April in Paris, France.

8. Author(s)/Editor(s)	9. Date
Various	March 1985
10. Author's/Editor's Address	**11. Pages**
Various	172

12. Distribution Statement This document is distributed in accordance with AGARD policies and regulations, which are outlined on the Outside Back Covers of all AGARD publications.

13. Keywords/Descriptors

Radio frequencies
Radiation effects
Radiology
Health physics
Safety engineering
Standards
Military operations
Military personnel

14. Abstract

Research conducted in the past few years provides a better understanding of the way radio-frequency radiation (RFR) is absorbed and distributed in living systems. This has led to general agreement on a common denominator for assessing the biological effects of RFR, which, in turn, has resulted in the development and application of new RFR safety guidelines. This lecture series will include presentations on

(1) the physical interactions of RFR fields with biological systems,
(2) the biological effects of RFR exposures,
(3) the measurement of RFR fields in operational settings, and
(4) the development and operational impact of new RFR safety guidelines.

This Lecture Series, sponsored by the Aerospace Medical Panel of AGARD, has been implemented by the Consultation and Exchange Programme of AGARD.

AGARD Lecture Series No.138
Advisory Group for Aerospace Research and Development, NATO
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Military operations
Military personnel

AGARD Lecture Series No.138
Advisory Group for Aerospace Research and Development, NATO
THE IMPACT OF PROPOSED RADIO FREQUENCY RADIATION STANDARDS ON MILITARY OPERATIONS
Published March 1985
172 pages

Research conducted in the past few years provides a better understanding of the way radiofrequency radiation (RFR) is absorbed and distributed in living systems. This has led to general agreement on a common denominator for assessing the biological effects of RFR, which, in turn, has resulted in the development and application of new RFR safety guidelines. This lecture series will include presentations on

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(1) the physical interactions of RFR fields with biological systems,
(2) the biological effects of RFR exposures
(3) the measurement of RFR fields in operational settings, and
(4) the development and operational impact on new RFR safety guidelines.

The material in this publication was assembled to support a Lecture Series under the sponsorship of the Aerospace Medical Panel and the Consultant and Exchange Programme of AGARD presented on 11—12 April 1985 in Rome, Italy, 15—16 April 1985 in Lisbon, Portugal and 18—19 April in Paris, France.

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